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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles related to periodontology that appeared during the year of 1945 are published in this year's issues.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided by contributing the material for this issue.

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"It is pleasant as well as profitable to turn, on fit occasions, from the bustle of active life to the study of the past—to the origin of our art, to the principles and necessities that called it into being, to the struggles of our ancestry. We are thereby better able to understand our own position, to know how far we have advanced, to whom we owe our progress, the labor still before us, and the places we ourselves are likely to occupy in the estimation of those who are to follow us."—John Watson

Quoted in Dental Chronology

See Review pp. 40 and 43

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-SIX

The New Concept of Periodontoclasia

By BERNHARD GOTTLIEB, M.D., *Dallas 1, Texas**

INTRODUCTION

EVOLUTION is peaceful development, the successive addition of new floors of knowledge to those which we have inherited from past generations. It is useful and comfortable to walk in this path and every peace loving human being does it. But if we discover that the first floor was constructed on untenable principles, reasonable people will not hesitate to tear down the building and start from scratch. Wise people will take pains to preserve every piece of artistic value for use in constructing a new building. We must not forget that our main task is not the tearing down but the replacing of the old building by a superior one.

In periodontia, the concept of the normal starting point of periodontal disease, the gingival sulcus, was a misconception. That was the misconstructed part of the first floor. The important role of the cementum, as the only tissue which maintains the connection of the tooth with the body, was not incorporated into the first floor of the building at all. In view of such major defects it may be advisable to recheck the building.

REATTACHMENT

Let us analyze the situation in the instance of reattachment. Until a quarter of a century ago it was the general belief that normally the base of the sulcus is at the cemento-enamel junction and its depth estimated at 1½ mm. It has since developed that the supposed "normal sulcus" does not exist. Everyone now knows that the cemento-enamel junction has nothing to do with the base of the sulcus. Examining millions of teeth, one cannot find a single tooth, where the base of the gingival sulcus is at the cemento-enamel junction all around the tooth. That base passes the junction at different points on the circumference of a tooth at *different times. It does not stay* at this junction longer than at any other place on the tooth surface. The epithelial attachment grows down along the cementum long before the base of the sulcus has reached the cemento-enamel junction. The shifting of the base apically goes on by progressive detachment of the epithelium from the surface of the tooth. That proceeds on the cementum surface the same way as on the enamel surface.

* Research Department, Baylor University, College of Dentistry.

Formerly there has existed the idea that pocket formation means detachment "of the periodontal membrane from the surface of the tooth". A well trained periodontist was supposed to procure "reattachment" in pyorrhea [periodontoclasia] cases under his care. It was understood that, that achievement meant to restore the base of the sulcus to its "normal position" at the cemento-enamel junction. As the concept of that "normal position" has been changed entirely, that of reattachment must be changed too. A pocket does not form by detachment of the periodontal membrane from the surface of the root. The periodontal membrane is the connective tissue between the root surface and the alveolar bone. The alveolar bone of a tooth together with its periodontal membrane is never present in the area of pocket formation. On the labial side of an anterior tooth the level of the alveolar margin is always apical to the deepest point of the epithelial attachment. In any pocket formation the epithelium grows at first along the cementum and detaches from the tooth surface at the base of the sulcus. The alveolar margin moves apically before the downgrowth of the epithelium. If the gingival margin follows the base of the sulcus, gingival recession results. If it does not, we are confronted with pocket formation.

Let us take a common example, a pocket on the labial side of an anterior tooth. The gingival margin did not follow the base of the sulcus because inflammation was present. Inflammation means tissue hypertrophy, following of the gingival margin means tissue atrophy. Where hypertrophy is present, atrophy cannot develop. Our first aim in treating a pyorrheic [periodontoclasia] condition is to remove the inflammation. If we succeed, atrophy develops, (we call it "shrinkage"), which had been prevented previously by the presence of inflammation. If there is little hope that the gingiva will shrink by conservative treatment, we may perform gingivectomy. It is not clear what could be reattached in such a case. As yet no one has had the idea of lifting the gingival margin in cases of gingival recession. It might be advisable to tear down completely the floor of reattachment in the old building. A single room will suffice in the new building, where we shall deal with the intrabony pocket.

An intrabony pocket is not an intra-alveolar pocket. The alveolar housing of the affected tooth is always gone in the area of pocket formation. An intrabony pocket is impossible on the labial side of an upper anterior tooth. If the alveolar bone is gone here, no bone is left. But on the lingual side of the same tooth, bone of the hard palate backs the alveolar bone proper. If the alveolar bone disappears from such an area, the palatal bone remains, causing the intrabony pocket. Gingival recession cannot develop here, because the palatal bone prevents the gingival margin from following the base of the sulcus, and thus prevents gingival recession. The same applies to any approximal surface, when the alveolar bone of one tooth has disappeared and the alveolar bone of the neighboring tooth is intact. This alveolar bone of the neighboring tooth is higher than the level of the base of the pocket of the affected tooth, in this way producing an intrabony pocket.

Fig. 1 may illustrate these conditions. It is an upper incisor. On the labial side (a) we see a deep pocket. The periodontal membrane (b) at the alveolar margin (c) is widened. The alveolar margin is apical to the base of the pocket (d). At the lingual side, the base of the pocket (e) is apical to the bone margin (f). The alveolar margin of this tooth is at (g), apical to the base of the pocket. The bone f-g is palatal bone, having nothing to do with the tooth. That is an intrabony pocket.

In such intrabony pockets reattachment can be ventured. All the epithelium in the pocket has to be removed, likewise the superficial layer of the root, being probably infected. Inflamed connective tissue (granulation tissue) must be removed also. The blood clot formed between the tooth and the bone opposite must be protected from being lost. That can be accomplished by connecting the labial with the lingual gingival margin by a suture. If the operation is a success, the blood clot is organized into connective tissue, a new cementum layer is deposited on the root surface, new bone forms opposite the tooth and both become connected by fibres. It is a similar

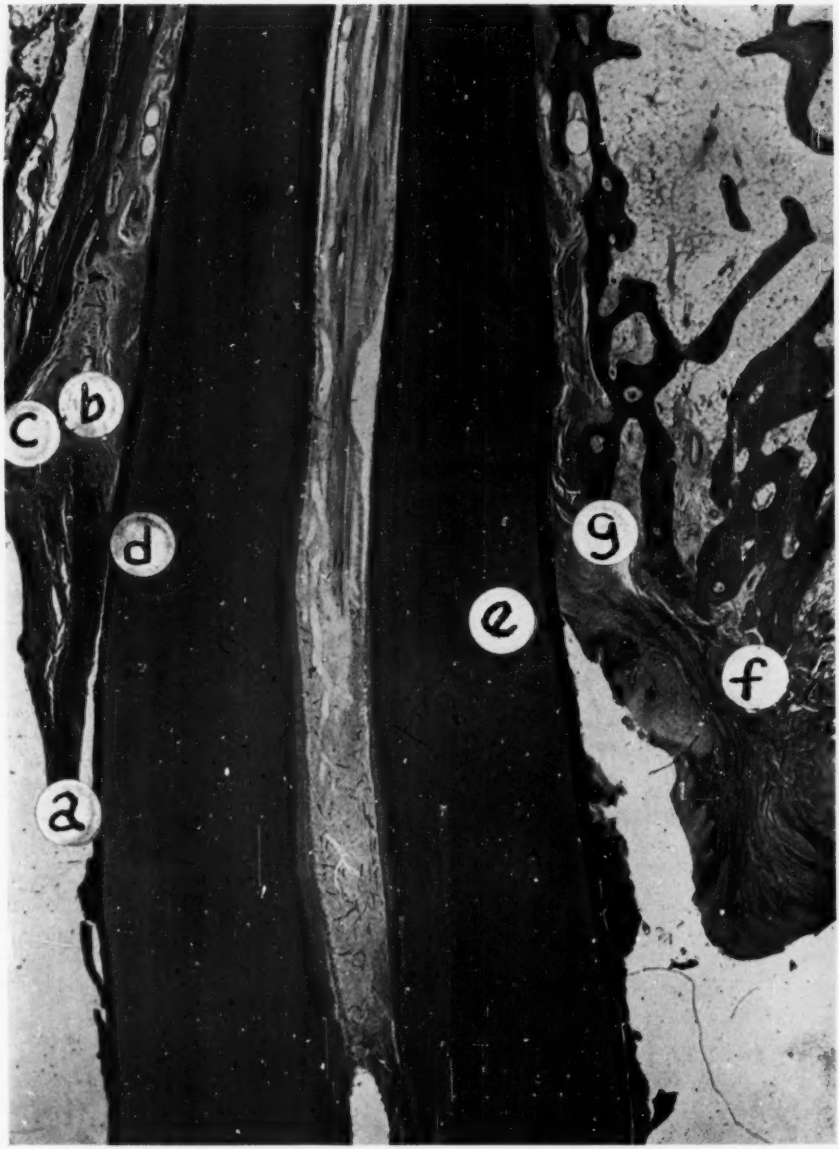

Fig. 1. Upper incisor. (a) Labial gingival margin; (b) widened periodontal membrane; (c) alveolar margin; (d) base of the pocket; (e) base of the lingual pocket; (f) bone margin; (g) alveolar margin.

process such as we are used to finding in repaired tooth resorptions. The new epithelial attachment terminates at the occlusal end of the new cementum deposition.

However, if reattachment is not planned and the tooth is going to be saved, it represents the only occasion, where bone removal to a level beyond the base of the pocket is justified. The reason given for removing bone in pyorrhea [periodontoclasia] cases, because of being "infected", seems to be a misunderstanding. Infected bone is only to be found in instances like osteomyelitis. In such cases, as in dry socket, this bone is severed from the healthy bone by resorption and is eliminated as a sequestrum. An infection of bone in pyorrhea [periodontoclasia] never occurs. Microörganisms do not live in living tissue. Only necrotic tissue is able to harbour microörganisms, as in the periodontal pocket as well as in the necrotic pulp and periapical tissue, and in the half necrotic deposits on the heart valves. Microörganisms may come occasionally into the blood stream, but are eliminated quickly under normal conditions. If somewhere near the circulation necrotic tissue is available, it attracts the microörganisms. Here they can multiply and a focus of infection develops. In the drained periodontal pocket, conditions for such a process are not favorable. The granulation tissue, which is found around a periodontal pocket is an inflammatory reaction to chronic irritation. Orban has shown, that one can remove the last traces of such inflammatory reaction by use of oxygen produced by superoxol application. Healthy connective tissue takes its place. We have a reason for saving every bit of bone at the alveolar margin. *Regeneration of bone here seems to be rare.*

The indiscriminate formation of flaps in treating pyorrhea [periodontoclasia], involves risks which should not be taken. Fish (London), in experimental flap operation on dogs, with consecutive histological examination, showed, that one always gets an accelerated elimination of the tooth, a shortening of the clinical root and a lengthening of the clinical crown. That means an acceleration of the process which we have to fight. A flap operation seems to be justified only in cases where reasons of surgical technique indicate it and the foreseen gain is greater than the involved risk.

POCKET FORMATION AND GINGIVAL RECESSION

Another example of misunderstanding is the relationship between gingival recession and pocket formation. Both have in common the speeded movement of the base of the sulcus apically. In gingival recession the gingival margin follows the base. That means that the depth of the sulcus remains the same. No one has ever observed inflammation or tartar formation in connection with gingival recession. Accordingly, no one has expressed the idea that the apical movement of the base of the sulcus here is caused by calculus or inflammation.

Let us take the commonly known instance of gingival recession on the labial side of an upper cuspid. If the apical movement of the base of the sulcus happens on the distal side of the same cuspid, instead of the labial side, the result could never be gingival recession. The gingival margin here is the tip of the papilla, common to the cuspid and the first premolar. Supposing that the first premolar is in a healthy condition, the tip of the papilla would be held firmly in its place by the epithelial attachment of this tooth. A following of this gingival margin along the movement of the base of the sulcus at the distal side of the cuspid is impossible. A pocket *must* develop in this place, no matter what the gingival conditions otherwise are. Once a pocket has developed, unhygienic conditions are established, tartar formation and inflammation are the natural consequences. The many cases of gingival recession observed after extracting a neighboring tooth may be, to some extent, explained that way. If the extracted tooth had remained in place, and healthy, a pocket would have developed in this place, instead of gingival recession.

The root surface looks at the beginning of pocket formation probably the same as in gingival recession: no calculus formation. The gingival tissue shows no inflammation. While the tooth surface in gingival recession can be cleaned, and no irritation develops, the denuded tooth surface in the pocket can neither be reached by the tooth brush nor by any natural cleaning device. Accumulation of soft deposits are unavoidable,

deposition of calcium salts into the soft deposits transforms them into tartar, the pressure of the soft tissue condensates the deposit, occasional bleeding stains it dark, tissue exudates are added, and we are confronted with the subgingival "serumal" calculus. Such deposits produce irritation of the adjacent gingiva and that is answered by inflammation. That in turn increases the volume of the gingiva, which is identical with farther increasing the depth of the pocket.

Fig. 2 seems to be suitable to illustrate these conditions. A deep pocket, with the base below (d), shows accumulation of exudate. The epithelium (e) has irregular growth with areas of increased inflammation of the connective tissue (f). The basal part of the root surface is free from deposits up to (c). Tartar formation (b) developed between the cemento-enamel junction (a) and (c). This tartar cannot be made responsible for the deepening of the pocket, not even for the inflammation at the base. That seems to be produced by the collection of exudate and other necrotic material. While the cemento-enamel junction is clearly defined at (a) we see only a dark line to the end of the illustration, which may be a thin layer of cementum. This poorly developed cementum may furnish the explanation for the probably poor attachment of the connective tissue, which gave occasion for the epithelium to move its attachment apically. If, however, the dark line is not cementum that epithelium was present from the time of dentin formation.

The question arises, can persistent inflammation induce the epithelial attachment to move apically? This may be answered in the following way: In the presence of a highly developed cementum barrier, nothing can cause downgrowth of the epithelium along the cementum. Even a surgically peeled off flap will reattach partly to the artificially denuded root surface. Such a reattachment may happen also to the enamel surface which points to differences in quality even of that tissue. In low resistance of the tooth surface, inflammation as

Fig. 2. (a) Cemento-enamel junction; (b) tartar; (c) end of tartar deposition; (d) exudate; (e) irregular growth of epithelium; (f) inflammation.

well as persistent occlusal trauma may be able to do additional harm.

The following finding in a lower premolar of a 19 year old human being may help in the understanding of these conditions. Serial sections were available. Fig. 3 shows the base of the sulcus lingually at (a), on the enamel. The gingival margin is at (b). The limitation of dissolved enamel (d) is indicated by the cuticle (c). The epithelial attachment stops at the cementum (e) which shows up as a black strip. The connective tissue under the epithelium is free of inflammation. The alveolar margin (f)

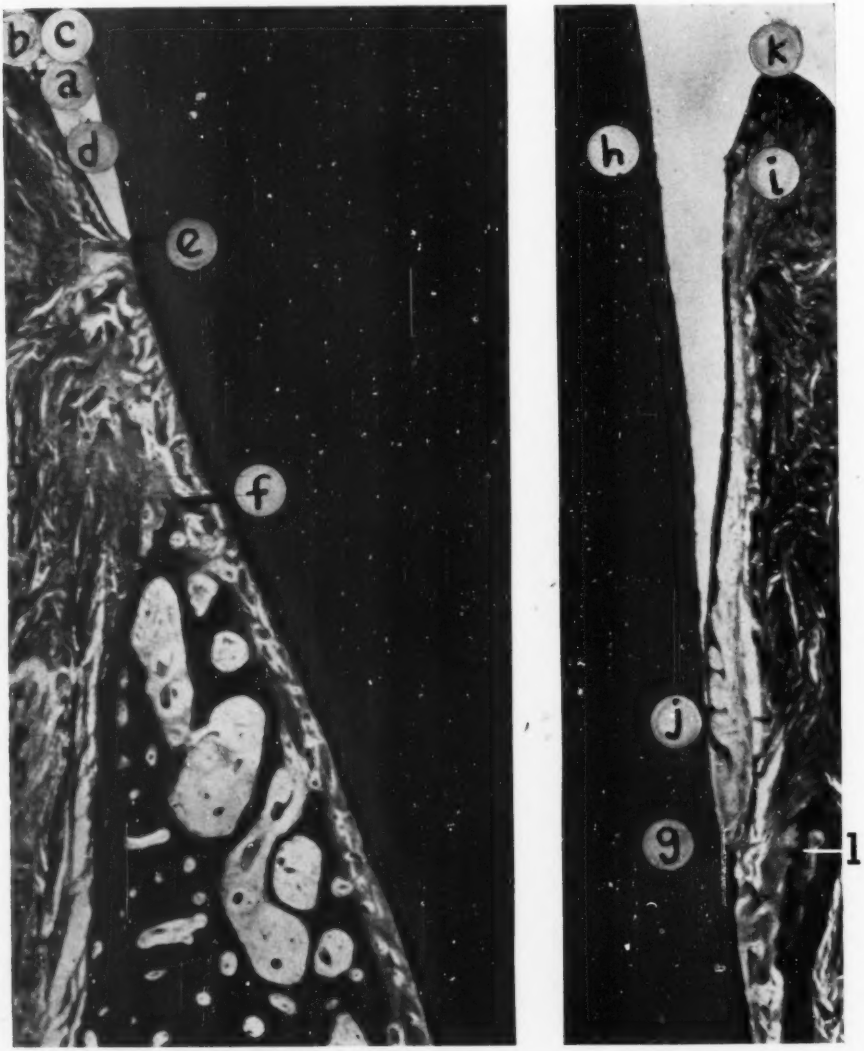

Fig. 3. Lower premolar of 19 year old human. (a) base of sulcus lingually; (b) gingival margin; (c) cuticle; (d) enamel lost by decalcification; (e) cemento-enamel junction; (f) alveolar margin; (g) Cementum on labial side; (h) cemento-enamel junction; (i) opposite end of epithelium; (j) base of pocket; (k) gingival margin.

appears to be more remote from the epithelium than we are used to seeing it in the average case. The conditions on the labial side are quite different. The cementum starts with a thickening at (g). It may be that a trace of cementum extends some distance coronally, but it could not be positively ascertained. Below this thickening the cementum equals that of the lingual side. At first we might be in doubt where the enamel ended here, before decalcification. The dentin surface which was covered by enamel is quite smooth. At (h) it starts to be rough. The opposite epithelium forms an angle (i) at that level, which appears to be the end of the enamel. Here we find no cuticle attached to the epithelium. That indicates that the epithelium was detached from the enamel surface before decalcification. It may be correct to assume, that the base of this sulcus was at (j) during lifetime. The roughness of the dentin surface between h-j is apparently the beginning of formation of deposits on the denuded surface. The gingival margin (k) did not follow the base. The alveolar margin (l) is below the epithelial attachment. This condition was only present in a few sections. The area of pocket formation was so narrow that the neighboring gingiva, being attached to the enamel kept the gingival margin at a high level preventing it from following the base. That is another topographical reason for pocket formation preventing development of gingival recession. In the usual instance the attachment to the neighboring tooth prevents the gingival margin from following the base.

The connective tissue is here free from inflammation, and the epithelium does not show any ulceration. The only fingerlike formations in the epithelium are around the base (j). That indicates that there was some chronic irritation present. We can imagine that unhygienic conditions were present also. The roughness of the root surface seems to be the start of tartar formation.

Fig. 4 shows a neighboring section of Fig. 3. The conditions on the lingual side are the same. On the labial side the enamel cuticle is still attached to the epithelium, showing the base of the sulcus at (a). Beginning at the enamel there runs a short strip of cementum. At (b) no cementum is present. Apically there is a smaller similar strip (c). During life the connective tissue at (b, c) and the opposite dentin were in contact with each other. They separated apparently during histological preparation. In the series a connection can be found between the cementless dentin area of Figs. 3 (b) and 4. The epithelium had not lined the outpocketing (b) of Fig. 4. This indicates that the pocket formation in Fig. 3 is of recent origin and that the separation of the epithelium from the enamel surface occurred a short time before death. We can imagine that additional deposits with stagnation of saliva and exudate could develop into a lateral abscess.

Similar anomalies of lack of cementum

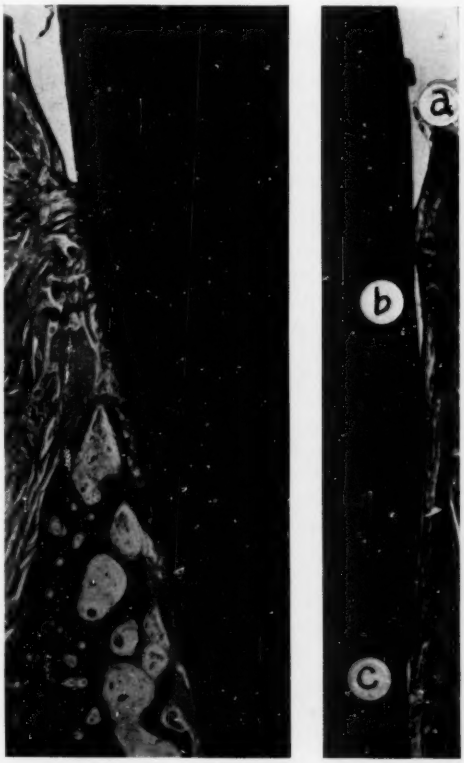

Fig. 4. (a) Base of sulcus; (b) and (c) cementum not formed.

formation on a smaller scale were observed previously.* In these cases the cementum did not start at the end of the enamel, but farther apically. The dentin between is covered by the extended epithelial attachment to the enamel. During development, the dentin, in the stage of its early formation, is covered by epithelium all the way, separating it from the surrounding connective tissue. The cementum-forming connective tissue breaks through that separation and brings the tooth into union with the surrounding connective tissue.

Nearly every dentist has seen children with a single denuded labial root surface of a single lower incisor due to gingival recession. The parents are alarmed without justification. The pathological background is the same as in Fig. 3. Here the anomaly affected only a narrow strip of the circumference. The gingival margin could not follow and a pocket developed. In the cases of gingival recession of lower incisors, recession can develop the anomaly affecting a more extended area. Below the lowered epithelial attachment a reliable cementum barrier may prevent farther movement of the epithelial attachment for a considerable time. In the gingival recessions mentioned it is mostly lack of cementum formation. In the other cases of gingival recession observed on the labial sides of upper cuspids or on the lingual sides of first molars, the continuous cementum deposition seems to be disturbed. These places are favorable for gingival recession. If the same happens in other places, a pocket develops.

We are accustomed to thinking of periodontal diseases of the kind which is discussed here, calling it "pyorrhea alveolaris" or "periodontoclasia" or any other name, as being connected with inflammation of the gingiva. Having made clear that the biological background of gingival recession and pocket formation are the same, we must realize, that the inflammation is *secondary*. That represents quite an alteration of the old "pyorrhea" building.

On the other hand there are inflammations with considerable hypertrophy of the gingiva, which develop *primarily* and have

Fig. 5. Lateral of a 19 year old person with isolated gingival hypertrophy. (a) Fibres inserting at the cementum hyperplasia, (b) epithelial attachment; (c) cell infiltration between fibre bundles; (d) dentin; (e) enamel space; (f) cementum hyperplasia.

* Fig. 26 in *Biology of the Teeth*. Gottlieb, Orban, Diamond. MacMillan, 1938.

no connection with pocket formation. Such an instance is the gingival hypertrophy in puberty. Many years ago such a case was published. It was gingival inflammation and hypertrophy on the labial side of an upper lateral of a 19 year old person. The gingiva covered the greater part of the tooth surface. In Fig. 5 we see the cemento-enamel junction of this tooth at the place where the gingival hypertrophy was greatest. A cementum hyperplasia (f) developed at the cemento-enamel junction, with strong fibres inserted (a). The epithelial attachment (b) ends at the occlusal border of the cementum hypertrophy. Some inflammation cells are to be found between strong bundles of fibres in the deeper parts (c).

At the lingual side of the same tooth and in other teeth of the same mouth, there was neither alteration of the gingiva nor cementum hyperplasia. At the lingual side of this tooth the epithelium had already started to grow along the cementum. In Fig. 5 we get the impression that a downgrowth of the epithelium may be prevented for a long time. Here we see that primary inflammation and hypertrophy may result in a strengthening of the cementum barrier. As such a cementum hyperplasia could not be found in other places of this mouth it appears justifiable to assume that it was caused by the gingival condition.

Therefore, we will need to differentiate between secondary inflammation, caused by pocket formation due to pathology of the cementum, and primary inflammation, which may increase the cementum barrier. Ziskin has helped to clarify the primary gingival inflammation, as being connected with disturbances in the internal secretions of the body.

THE CLINICAL SIGNIFICANCE OF THE EXTENT OF THE EPITHELIAL ATTACHMENT

The decisive step in developing denudation of the surface of the anatomical root (either in gingival recession or pocket formation) is the loss of attachment of the tissue to the tooth surface. The epithelial attachment grows apically, covering that part of the root surface, from which the connective tissue has been detached. The epithelial attachment separates from the tooth surface on its coronal end.

The mechanism of this process is not quite understood, but the fact is established. We figure that the deterioration of the quality of the tooth surface passes different grades. The attachment of the connective tissue fibres needs a high vitality of the tooth surface, the ideal being the non-calcified cementum. The quality may be lowered below the requirements of the connective tissue but suffice for epithelial attachment. If the deterioration of the vitality progresses, a detachment of the epithelium takes place and the part of the tooth surface concerned is eliminated entirely. Up to the present time it could not be ascertained of what these changes in quality consist. But we need such a supposition in order to explain actual findings.

In view of this concept we may assume that the extent of the epithelial attachment varies widely. If a strong cementum barrier exists for a considerable time and the detachment of the epithelium goes on, the length of the attachment is reduced. In such cases we may find exceedingly short epithelial attachments. In other cases we may find long epithelial attachments. They indicate that during a period of time a considerable area of the root surface was overgrown by epithelium without corresponding detachment at the coronal end.

Fig. 6 illustrates such a condition. At an approximal papilla (a) there is some tartar deposited in epithelial ulcers. In the deeper parts of the connective tissue there are pieces of necrotic material (1), apparently remnants of bleeding due to some trauma. A case history was not available. A clinical examination would not have revealed more than existing unhygienic conditions without pocket formation. Radiographs would reveal a broadening of the periodontal membrane on the right side. The base of the sulcus is on both sides at the cementum (m). The epithelial attachment (b) at the tooth (h) is an unusually long one. It reaches down near to the resorption (c). Fig. 7 shows that condition in a higher magnification. The tooth resorption is repaired to a great extent by bone deposition (d). The alveolar margin

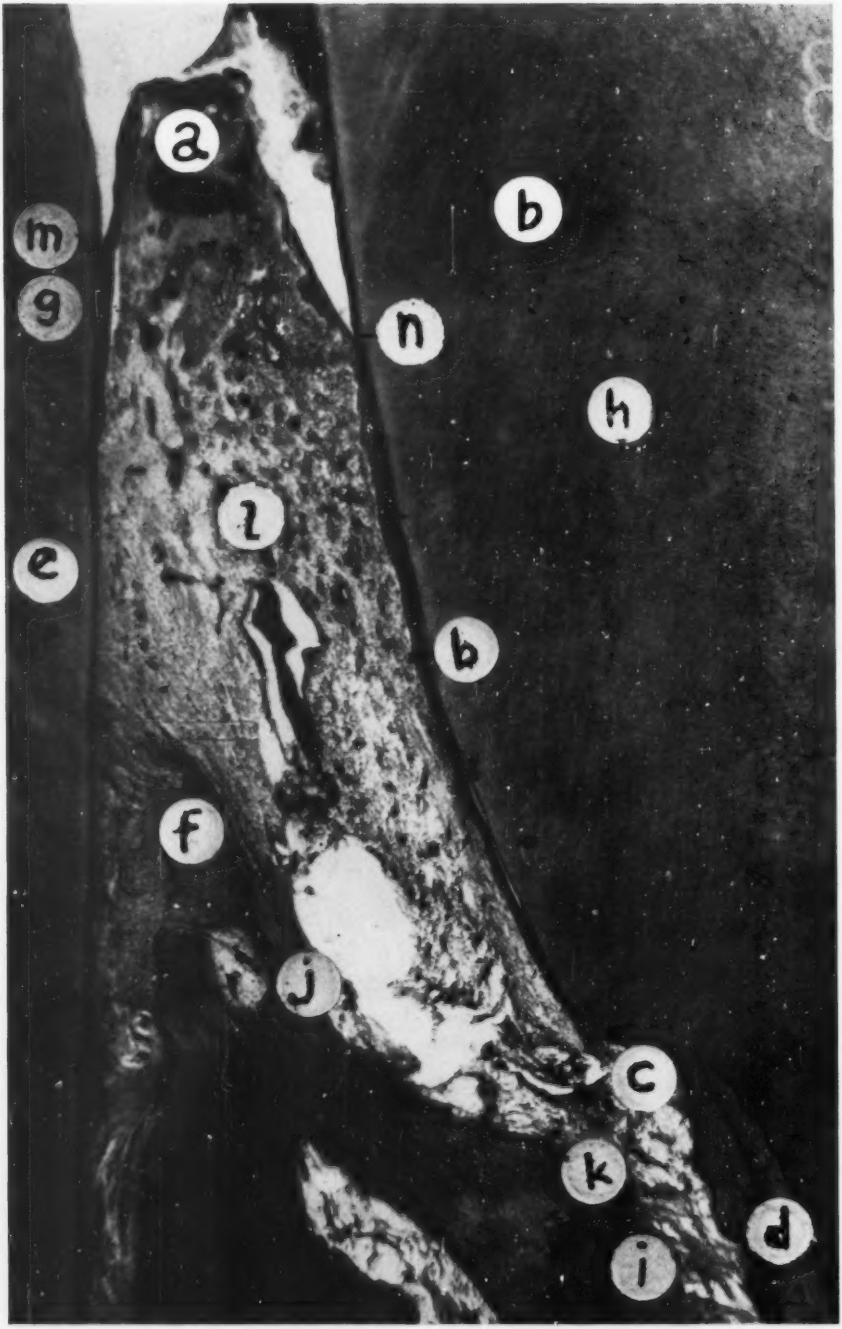

Fig. 6. Interdental papilla. (a) Necrotic material on ulcer; (b) and (g) epithelial attachments; (c) resorption of tooth (h); mostly repaired (d); (f) alveolar margin of tooth (e); (i) alveolar bone; (k) alveolar margin of tooth (h); (n) base of the pocket.

(f) of the tooth (e) is perhaps farther from its epithelial attachment (g) than we usually find it. Otherwise the conditions at that tooth appear normal. The alveolar margin (f) is not the alveolar margin of the tooth (h). We would suggest that it is at (k). The bone (j) cannot be considered as the alveolar bone of the tooth (h) being separated from it by an epithelial layer. It is exclusively alveolar bone of the tooth (e). It is only for topographical reasons that this bone is situated opposite the tooth (h). A portion of a root, which is lined by epithelium cannot maintain alveolar bone any more.

Corresponding to the repair of tooth resorption (d) the opposite bone received deposition also (i). At the time when the tooth resorption started the opposite bone could not have reached as near to the tooth (h) as it does now. It would have collided with the original tooth surface. At the time of the culmination of the resorption, the bone reached probably to the level (j). When the reparation (d) of the tooth resorption started, the bone grew nearer to the tooth until it reached the normal periodontal distance to the new concave tooth surface. That is the histological finding.

CLINICAL LANGUAGE DESCRIPTION OF FINDINGS

The resorption, as well as the long epithelial attachment point to the probability that the root surface was damaged somehow, in the past. The damage was answered by the connective tissue in one way and by the epithelium in a different way. The connective tissue performed resorption. The fact that the resorption is repaired to a great extent indicates that the damaging factor was no longer active, some time before death. It was probably of short duration like a single trauma. Any resorbed area of a tooth can be repaired without leaving permanent damage. The tooth resorptions in the course of orthodontic treatment are an example of this.

The reaction of the epithelium is quite different. Continuous deposition of new cementum layers serving as a barrier, has apparently ceased, in connection with the trauma, and made it possible for the epithelial attachment to grow down. That means that the affected part of the tooth was eliminated from connection with the connective tissue. Such a condition cannot be repaired by nature. That is the important difference between resorption by connective tissue and overgrowth by epithelium. A detachment of such an overgrowth of the epithelium from the tooth surface means gingival recession or pocket formation. It can happen in quite a short time. We can observe such things developing "over night."

We can predict that gingival recession is impossible in this case for the gingival margin is held by the tooth, Fig. 6 (e). When the epithelial attachment separates from the tooth (h) a pocket must form. In the deeper parts it will be an intrabony one, on account of topographical conditions. Thus we might realize that growing down of an epithelial attachment, caused by lack of an effective cementum barrier, may be followed by *gingival recession, simple pocket formation, or formation of an intrabony pocket.* The biologic principle is in all cases the same. The result depends entirely on the topographic conditions of the area involved.

Fig. 7. Higher magnification of the lowest part of the epithelial attachment (b) from Fig. 6.

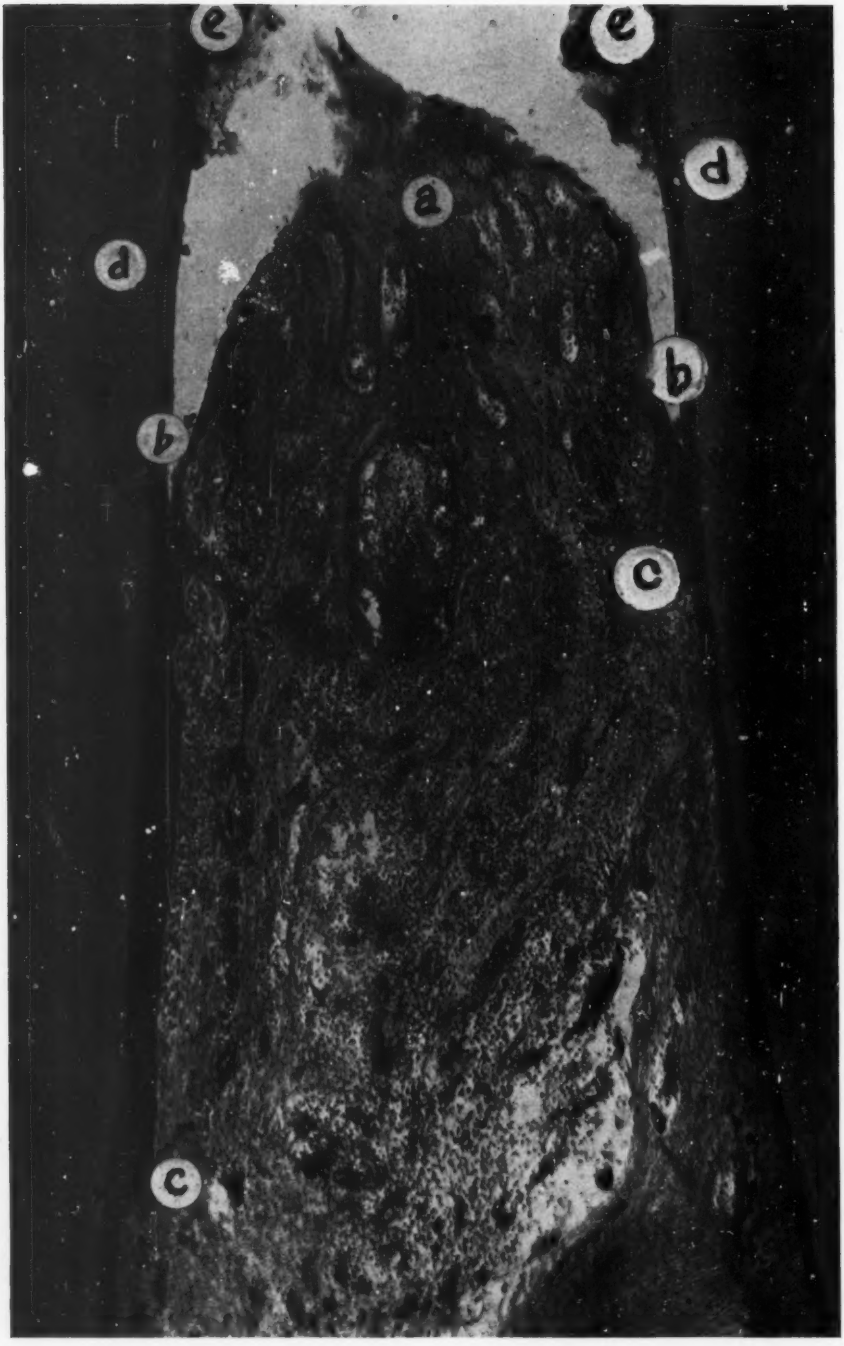

Fig. 8. (a) Interdental papilla; (b) and (b) base of sulci; (c) and (c) ends of epithelial attachment; (d) cementum; (e) tartar.

We may find long epithelial attachments without any sign of injury. In Fig. 8 we see a medium sized inflammation in an interdental papilla. The base of the sulcus on both sides is at the cementum (b). On the right side the epithelial attachment is of the usual extent. It ends at (c). On the left side it ends at (c). The inflammation decreased to zero and cannot be made responsible for the downgrowth of the epithelium. Apparently the cementum barrier failed to act here. The detachment of such an epithelial attachment may be only a matter of hours and a shallow sulcus becomes a deep pocket. Perhaps more striking is Fig. 9. The base of the sulcus is at the cementum (b); the sulcus is very shallow. The epithelial attachment (d) reaches to (c). The retro-epithelial connective tissue shows practically no inflammation. No fingerlike extensions of the epithelium indicate irritation. Clinically this case may be called ideal. The alveolar bone is below the epithelial attachment, not visible in the illustration. Suddenly a far reaching detachment may develop. Contrary to Figs. 6 and 8 such a detachment will result in gingival recession, if the area is broad enough and not narrow like in Fig. 3.

The length of the epithelial attachment can not be ascertained either by clinical examination or by the radiograph. We know that we cannot examine properly the labial or lingual side of a tooth by the radiograph. In approximal spaces we may get some indications of a long epithelial attachment. In a case where the neighboring tooth is present, the sulcus is shallow, and the periodontal membrane is broadened, it may be suspicious (Fig. 6). The same will apply to any increased distance between the base of the sulcus and the alveolar margin. Fig. 10 may show that such a finding does not always indicate a lengthening of the epithelial attachment. The base here is at (a). The epithelial attachment is short, despite the inflammation and ulceration of the epithelium in the sulcus. The distance of the epithelium to the alveolar margin (b) is considerable. Several cementum resorptions (c) suggest a low quality of the cementum surface. The cementum below the epithelial attachment was apparently sufficient barrier against downgrowth, but may not have sufficed for maintaining an alveolar bone. In some cases tilting of the tooth may be a cause for undue resorption at the alveolar margin.

Fig. 9. (a) Cementum; (b) base of sulcus; (c) end of the epithelial attachment.

The following illustrations are intended to increase the understanding of these problems. Fig. 11 shows the epithelial attachment to the root of a pig's tooth. At first it looks like an epithelial attachment at the cemento-enamel junction. The base of the sulcus is at (a). The cuticle of the root (b) is detached from the root surface but it remained in connection with the epithelium. The peripheral parts of the epithelial attachment apparently shrunk away from the tooth surface during histological preparation. That part of the attachment was probably not firmly attached to the tooth any more. The shrinkage produced the empty space (d) in the section, which looks like a place where enamel was dissolved by acid. The rest of the epithelial attachment (c) was still firmly attached to the cementum. Below that epithelium we see a strip of non-calcified cementum. This illustration demonstrates how the peripheral portion of the epithelial attachment may have prepared its separation from the tooth surface to a degree that shrinkage during histological preparation was enough to complete the separation. The epithelium took along with it the already formed cuticle. The apical part was still firmly attached to the cementum and remained so.

Fig. 12 shows the epithelial attachment to a rat's molar. The cementum hyperplasia at the cemento-enamel junction (a) prevented, apparently, the downgrowth of the epithelium for a considerable time. The length of the epithelial attachment was eventually reduced. The epithelium of the sulcus is bulging into the connective tissue (b) probably due to food impaction. The black material at (e) cannot be explained by the writer. It is a place which is normally occupied by enamel and empty in decalcified sections. It may be that it is carious enamel. At (d) and more clearly shown at (d^1) there is a thin layer of non-calcified cementum, the best barrier against downgrowth of epithelium.

WHAT CAUSES DOWNGROWTH OF EPITHELIUM?

We have been uncertain as to the cause of downgrowth of the epithelium. The tooth forms a hole in the epithelium. The epithelial attachment is the epithelium surrounding the hole. Wherever such a hole exists in the epithelium, the surrounding epithelial cells strive to close it in order to keep the epithelial blanket of the body intact. When we extract a tooth, that hole is closed in a short time.

We must look at this problem from a different angle. *What prevents the epithelium from growing down towards the closing hole?* The epithelium does not need stimulation, it must be prevented from grow-

Fig. 10. (a) Base of sulcus; (b) alveolar margin; (c) resorptions of cementum; (d) cemento-enamel junction; (e) tartar.

ing down. The continuous deposition of new cementum inside the epithelial attachment forms the barrier, over which epithelium cannot grow. When such a cementum layer becomes calcified and old, without getting a new layer deposited on top, the barrier stops being effective and the epithelium grows down apically. It may encounter a new barrier and stop there or it may succeed in closing the hole, eliminating the tooth.

WHAT CAUSES THE ALVEOLAR BONE TO DISAPPEAR?

The formation and maintenance of the alveolar bone proper as a housing of the tooth contains some unsolved problems. But some facts are certain: it forms with the tooth; it transforms according to the changes on tooth surface due to resorptions (Fig. 6); the shape of the alveolar bone follows any tooth movement (orthodontics); it disappears when we remove the tooth. In the latter case the transformation of the bone concerned follows the needs of the remaining components in this area. In cases of bone loss in pyorrhea alveolaris [periodontoclasia], the presence of the tooth may prevent such transformation. Where cementum is not formed (Fig. 3), the corresponding alveolar bone is not formed either. We do not need irritation to make alveolar bone disappear. On the contrary, we need stimulation in order to make it form and persist. We are inclined to believe that no other tissue furnishes that stimulus, but cementum. It is quite a temptation to argue that if the cementum does not hold any more fibres, the opposite bone becomes useless and disappears. But that seems not to be the whole story. In some teeth without function the alveolar bone persists for many years while no connecting fibres exist, only loose connective tissue filling the periodontal space.

NOMENCLATURE

A book on this subject was published in 1933 (Second Edition, 1936) under the title *Gingival Inflammation and Loosening of The Teeth.** The first paragraph of the introduction reads: "The discussion of this subject in the past decade was characterized by that of names. We choose the simple words in the title in order to indicate that we are at this time interested in clearing up the subject itself."

It appeared advisable to allow the subject to ripen. A quarter of a century may be enough,

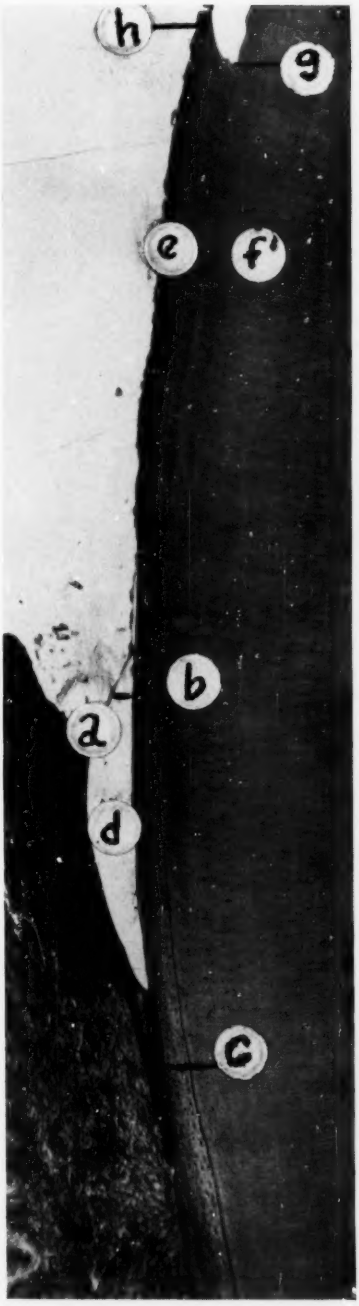

Fig. 11. Tooth of pig. (a) Base of sulcus; (b) cuticle; (c) fixed part of epithelial attachment; (d) place produced by shrinkage of the loosened part of the epithelial attachment; (e) cementum; (f) dentin; (g) cemento-enamel junction; (h) cementum spur, common in pigs.

* Gottlieb and Orban, Berlinische Verlagsanstalt, Berlin.

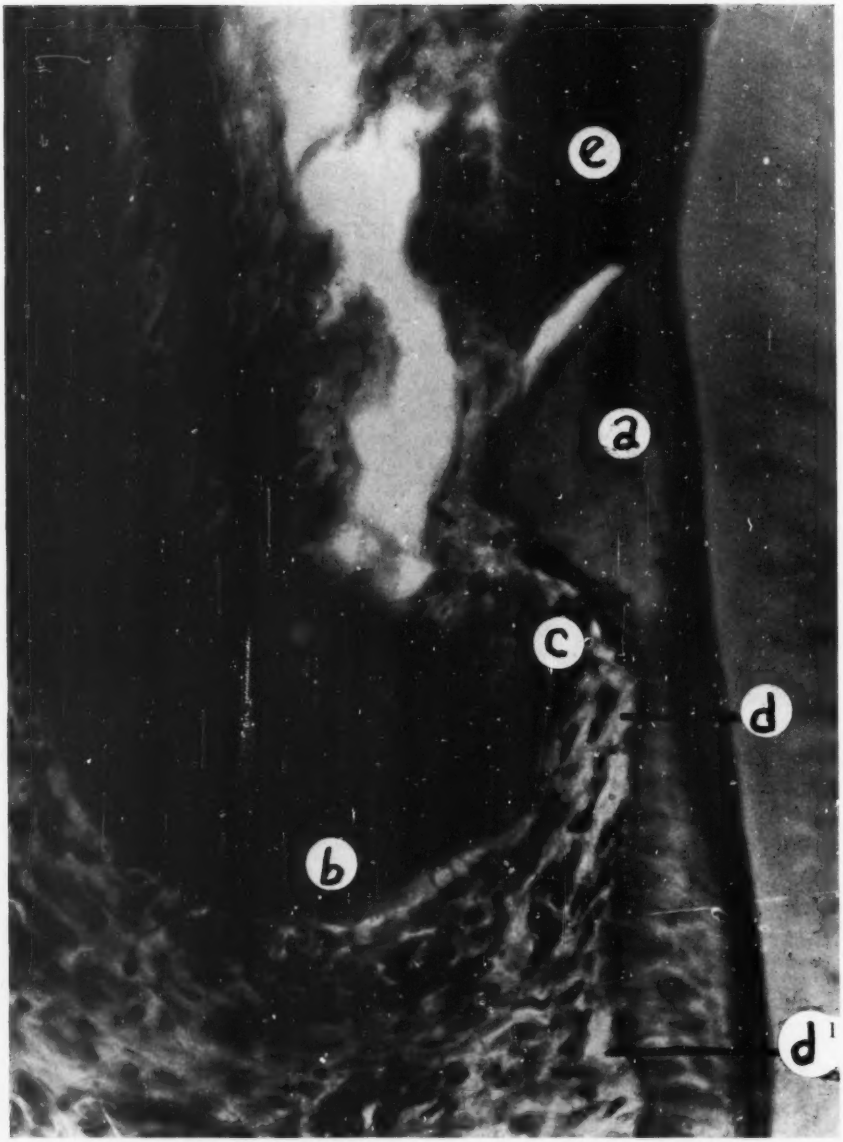

Fig. 12. Molar of a rat. (a) Cementum hyperplasia; (b) bulging of epithelium; (c) short epithelial attachment; (d) and (d^1) uncalcified cementum; (e) necrotic material.

that being the time that elapsed since the publication of the "Epithelial Attachment".* Feeling now, that a healthy baby has been delivered, it may be justifiable to give it a proper name, corresponding to the available details. A name without a baby is no good, but a baby without a name gives a lot of trouble. At first we will need to make a rearrangement of the material. We must take in gingival recession, having the same biological background as pocket formation. On the other hand we will have to loosen the ties with gingival inflammation. There are some gingival inflammations, which produce a beneficial effect on the cementum (Fig. 5). In other instances we find pocket formation without inflammation (Fig. 3), at least in the beginning. In a few instances, inflammation may deepen the pocket, producing downgrowth of the epithelium.

The periodontal problems under discussion, are based on a pathological condition of the cementum. Accordingly such conditions may be called *cementopathia.* This name would cover gingival recession as well as pocket formation and pathological wandering of the teeth. Right in the beginning of the era of the new periodontal concept, nearly a quarter of a century ago, it was shown, that pockets or gingival recession may develop in cases of wandering, as a rule, on the side wherefrom the tooth moved, not on the opposite side. That means that the affected cementum area has reached the epithelial attachment.

Gingival recession and pocket formation are produced by pathology of the cementum adjoining the epithelial attachment. It may be advisable to connect both under the heading *marginal cementopathia,* producing either *gingival recession or pocket formation.* The pocket may be formed by soft tissue from the base to gingival margin. Such a pocket may be called *Simple Pocket.* The bone margin is then apical from the base. If the bone margin is between the base and the gingival margin we are confronted with an *intrabony pocket.* The soft tissue forming the pocket may be *with or without inflammation or suppuration.*

*D. Mo. Z. 39:142. 1921.

If, however, the affected cementum area is located far from the epithelial attachment the marginal structures show normal conditions in the beginning, and *wandering* is the only clinical symptom. We called it temporarily: diffuse atrophy. We propose to call it now *deep cementopathia (cementopathia profunda).*

In gingival inflammation we will have to differentiate between *primary* and *secondary gingival inflammation.* The *primary* is not caused by pocket formation but primarily by some systemic condition (for example puberty, pregnancy). The *secondary* one is caused by the unhygienic conditions produced by pocket formation.

The idea that other pathological conditions of the supporting structures should be connected with *cementopathia,* making a common nomenclature, may not be advisable. It should be clearly understood, that the clinical and pathological picture of *cementopathia* is a well defined entity.

CONCLUSION

Some systemic institution is apparently responsible for the continuous cementum deposition. If a disturbance of that mechanism goes on for a considerable time, the epithelium succeeds in closing the hole, that means the teeth are eliminated. The person is then not any more eligible to become a patient of a periodontist, being rather taken care of by a prosthodontist. Only those people are patients of periodontists, who have had a cementum disturbance of relatively short duration. Irregular downgrowth of the epithelium may have caused pocket formation. Cementum pathology in the deeper parts may have caused wandering. If such a condition is not treated, it cannot heal and is bound to progress. If properly treated, full success will be achieved.

We hope to have succeeded in showing that pathology helps to better understand clinical facts. The clinician should not be supposed to learn extensive nomenclature believing that it will be beneficial. *Understanding of the underlying facts is of main importance.*

Studies In Dental Nomenclature: A Series

IV. A CLASSIFICATION OF PERIODONTAL DISEASES

By HARRY LYONS, D.D.S., *Richmond 19, Virginia*

IN THE PREVIOUS articles of this series, the development of terms useful in the realm of periodontology was discussed. It appears now that a classification of periodontal diseases employing the recommended terms might be valuable in teaching and in an approach to considerations of diagnosis and treatment.

Many classifications of the periodontal diseases are presented in the literature. Bunting (1), Hill (2), Merritt (3), Orban (4), Stillman and McCall (5), Thoma (6), and others have offered classifications based upon varying ideas and approaches. An evaluating comparison of these is difficult because of differences in the nomenclature employed and variations in the basic principles upon which these workers developed their classifications. Efforts have been made to devise classifications of the periodontal diseases on the basis of etiology. The great voids in our knowledge of the causes of periodontal disease make such an approach obviously limited in value. Other attempts at classifications were based upon symptoms. The wide variations in the symptoms presented by any given disease in its course limit the usefulness of this plan. The logical basis for a classification of the diseases which occur in this area would appear to rest on the diseases themselves—the basic reactions and changes in the involved tissues, about which present knowledge is quite extensive.

All acquired diseases (as distinguished from inherited tendency disorders) may be divided into three general types, depending upon the character of the basic pathologic processes. Acquired diseases are either *inflammatory*, *degenerative* or *neoplastic* in general character. Since the periodontal diseases are acquired disorders, they also lend themselves to similar consideration. The three general types, while distinct in their incipient stages and basic character, often tend to merge with each other or to be complicated, one by another, in later stages of a given disease. For example, a long-standing, chronic *inflammatory* process may ultimately lead to an inflammatory new growth or granuloma and so give rise, secondarily, to a *neoplastic* process. The whole process, however, is *inflammatory* in origin and character. Conversely, a basically *neoplastic* disease such as carcinoma may be secondarily involved by an infectious, *inflammatory* process leading to ulceration. However, the whole process, by origin and character, is basically neoplastic. These observations are important for many reasons, among them being considerations of therapy. Here are two examples of disease processes both characterized by *inflammation* and *neoplasia*—but requiring entirely different therapeutic considerations. In the first instance, treatment should be *primarily* directed at the basic inflammatory process and its cause, with *secondary* attention to its *resultant* neoplastic sequela. In the second instance, the reverse order should prevail. Comparable situations can be cited

wherein *inflammatory* and *degenerative* processes merge or complicate each other in later stages. These principles prevail in full force in the field of periodontology. The failure to recognize and to think in terms of these principles may account for many unsatisfactory results. Good practice and teaching demand that cases be considered in the light of their *primary* natures and not in terms of *secondary* complications which often highlight a case clinically in later stages. It is with this in mind that the following classification is offered and for which no originality is claimed.

In an explanatory discussion of this classification of the periodontal diseases, it may be observed that the three major divisions of the classification are noted as "Inflammatory," "Degenerative" and "Neoplastic." Efforts to increase the number of major divisions are without foundation in principles of pathology. For example, the creation of a major section on periodontal atrophy is unwarranted since atrophy may be considered a type of *degenerative* process in its broadest sense. Similarly, traumatism cannot be logically set up as an independent section since traumatism (the effects of trauma) is either inflammatory or degenerative in character, depending on the intensity and the duration of the traumatic factor.

In naming the diseases in the three major divisions, reference is made to principles discussed in the previous articles in this series. All *inflammatory* diseases may be designated by the use of the suffix "-itis." Inflammatory disease of the gingiva is, therefore, designated "gingivitis" and inflammatory disease involving portions of the periodontium (gingiva, periodontal membrane and alveolar bone) is accordingly designated as "periodontitis." As noted in the classification, the severity of gingivitis may vary from "acute" to "chronic," this being also true of all the types of periodontitis. The term "sub-acute" may also be useful in describing the severity of the inflammatory diseases of this field. Inflammatory diseases of the gingiva and periodontium also vary widely in the nature of their exudates, tissue changes, and end results. Accordingly, these diseases are also described as "simple," "edematous," "pigmented" (metallic), "hemorrhagic," "purulent," "ulcerative," "necrotizing" (ulcero-membranous, Vincent's, etc.), "hyperplastic," and "desquamative." No effort is made in this classification to identify these diseases with their causative agents. While this may be done in certain instances, the causes of most cases are either multiple, vague or altogether unknown. In the sub-section on "Periodontitis," the term "simplex" is used to designate those cases developing from previously prevailing gingivitis and the term "complex" to designate the much more involved cases of periodontitis developing secondarily to previously prevailing degenerative disorders of the periodontium.

The division of degenerative diseases of the periodontium includes all the degenerative processes of the structures supporting and surrounding the teeth, irrespective of cause or causes. They are collectively designated as "periodontosis." To use this term for any one degenerative disease is to arbitrarily restrict its use. Some of the periodontal degenerations are senile atrophies; others are atrophies of disuse; still others *follow* prolonged trauma or inflammation, or

A CLASSIFICATION OF PERIODONTAL DISEASES

I. Inflammatory				II. Degenerative	III. Neoplastic	
GINGIVITIS		PERIODONTITIS		PERIODONTOSIS	TUMORS OF THE GINGIVA AND PERIODONTIUM	
Acute	*Chronic*	*Simplex*	*Complex*		*Benign*	*Malignant*
Simple Edematous Hemorrhagic Purulent (abscess) Ulcerative Necrotizing (ulcero-membranous, etc.) Desquamative	Simple Edematous Pigmented (metallic) Hemorrhagic Purulent Ulcerative Necrotizing (ulcero-membranous, etc.) Hyperplastic Desquamative	Secondary to Gingivitis (all types)	Secondary to the degenerative disorders or severe trauma of the periodontium The inflammatory complications include all types noted under gingivitis	Atrophic Periodontosis (Periodontal Atrophy) 1. Senile 2. Presenile Traumatic Periodontosis (Periodontal degeneration resulting from hyper-functional stresses) Periodontosis Gravis (Periodontal degenerations of systemic origins, associated with the deficiency diseases, endocrinopathies, systemic toxicities, blood dyscrasies, metabolic diseases or unknown etiologic factors)	Gingival or Periodontal: Fibromatosis (Elephantiasis) Granuloma Giant Cell Tumor Fibroma Fibro-osteoma Papilloma Lipoma	Gingival or Periodontal: Carcinoma Melanoma Fibrosarcoma

develop as a result of numerous abnormal systemic states, such as the deficiency diseases, toxicities, endocrinopathies, etc. There is much yet to be learned of these latter relationships. Present knowledge in this field is not yet wide enough to justify an attempt to do much more than indicate the general character of the periodontal degenerations. Unfortunately, most of these cases are not presented for care until *secondarily* complicated by inflammatory processes, at which stage they are designated "periodontitis complex." The importance of such a classification as this one may be exemplified here again. To treat cases of this type as *primary* inflammatory involvements is to miss the main pathologic features of the case and can result only in limited improvement if not complete failure.

Unfortunately, tumors occur in the periodontal field and, as elsewhere in the body, both benign and malignant types occur in this area. As previously discussed, some of these neoplastic processes are inflammatory in origin; others are secondarily complicated by inflammatory processes. The need for thorough diagnostic studies in these cases cannot be emphasized too strongly. Treatment may be beyond the scope of many dentists, physicians or surgeons. Diagnostic consideration is the responsibility of all who are engaged in the field of oral health.

It is hoped that this classification and the discussions of nomenclature problems which preceded it in this series will help students and practitioners in the development of a rational concept of these important problems.

(Concluded)

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Nutrition and Dental Health

V. VITAMIN C.

By DOROTHEA F. RADUSCH, D.D.S., B.S., M.A., *Minneapolis 2, Minnesota*

IT IS generally agreed that severe gingivitis occurs in scurvy. In recent years it has been frequently claimed that gingivitis alone is indicative of vitamin C deficiency, and that it is commonly found without any other sign. This, in spite of data from well known sources, clinical and experimental, which show that no changes in the condition of the gums were noticed until after petechiae had appeared on the legs.

This subject, "Ascorbic Acid and the Health of the Gums" is discussed in *Nutrition Reviews* 3: 44-46, Feb. 1945. It is herein pointed out that in 1772, James Lind wrote in his *Treatise on Scurvy* that at the same time that the gums swell and bleed, the skin "is found covered with several reddish, bluish, or more frequently black and livid spots, equal with the surface of the skin, resembling

an effusion of blood under it. They are to be seen chiefly on the legs and thighs."

The review continues:

Similarly, the late distinguished clinician Alfred Hess pointed out: "The earliest sign of scurvy is usually a change in the complexion of an individual. . . . About the same time the patient loses his accustomed vigor, seemingly becomes indolent and complains of tiring quickly, and of breathlessness. . . . Very soon the gums become sore, bleed readily, and are found to be congested, spongy, and somewhat hemorrhagic at their edges. Absolute reliance must not, however, be placed on this sign for early diagnosis, as at times it does not appear until later. Careful examination at this stage will disclose petechial spots on the body, more especially on the legs." (*Scurvy, Past and Present*, Phil. pp. 176-77, 1920.)

This clinical experience was duplicated experimentally in the work of Crandon, Lund, and Dill (*N. Eng. J. Med.* 223: 353, 1940) when human scurvy was produced experimentally under carefully controlled conditions. No change in the condition of the gums was noticed until after petechiae had appeared on the legs, following six months subsistence on a virtually vitamin C-free diet.

Burrill (*J. D. Res.* 21: 353, 1942), after studying blood plasma levels of nearly 1400 dental cases, concluded that although a large proportion of cases with low plasma level had gingivitis, no causal relationship between low vitamin C and presence of gingivitis was apparent.

The question of the relationship of ascorbic acid to gingivitis was studied also by Stamm, Macrae, and Yudkin (*Brit. Med. J.* 2: 239, 1944) in three carefully controlled experiments involving 2961 members of the personnel of four Royal Air Force Stations.

Quoting from the summary in *Nutrition Reviews:*

In these experiments all available personnel at the stations were examined, except those who were already taking vitamin C or undergoing treatment for acute ulcerative gingivo-stomatitis. One experiment was made in the fall, and two in the spring. The gums of the lower jaw were examined at the outset for bleeding after digital massage; the presence or absence of bleeding was noted, and if present it was classified in one of three degrees of severity. Of those found with bleeding gums, alternate subjects were treated with daily doses of ascorbic acid for 3 weeks, (the doses varied from 100 mg. to 200 mg. daily at different times in different experiments) or with a placebo flavored with tartaric acid. The subjects did not know which they received. At the end of the period, examination of the gums was repeated by an observer who had no notes of the condition at first examination and no knowledge of treatment given. No significant difference was noted in the percentage of subjects improved in the two groups. In the third experiment three examinations were made at intervals of three weeks, and it was observed that despite treatment the number of the 600 subjects who showed bleeding of the gums varied only from 111 to 122, but included different individuals each time. Thus 48 subjects bleeding at first examination did not bleed at the second examination but 37 subjects then bled for the first time: at the third examination 34 more had stopped bleeding, while 41 had started.

The average daily ascorbic acid intake of the subjects was determined by measuring the weight of each food item served in the messes through one week, and titrating each foodstuff with Tillman's reagent. It was found to be 25.8 mg. in the fall and 16.8 mg. in the spring.

The authors concluded that there is a large random variation in the degree of bleeding of the gums, irrespective of treatment; that a similar incidence occurred at each of the four stations in spring and fall; and that no greater improvement was obtained by treatment with ascorbic acid than with control tablets.

Campbell and Cook *(Brit. Med. J.* 1: 360, 1941) had earlier reported that upon giving ascorbic acid to 14 patients whose gums bled promptly upon the application of pressure, a return to normal was noted in most instances in about four days. With this rapid healing there is reason to believe that the cure was brought about by the specific therapy.

If this is so, then one is justified in concluding:

a) that a therapeutic trial will serve to distinguish within a few days whether a given case of gingivitis is or is not due to vitamin C deficiency,

b) that there is no justification for ascribing gingivitis to vitamin C deficiency unless therapeutic trial with ascorbic acid rapidly cures it.

VITAMIN C AND WOUND HEALING

A number of experiments have demonstrated that both hard and soft tissues require vitamin C for ready healing. Thus the subject is a pertinent one for dentists.

As usual, most of the data result from experimentation upon laboratory animals. Ungar (*Nature* 149: 637, 1942) performed a series of tests on the effect of immediate injections of ascorbic acid into traumatized guinea pigs and rats.

This author established the amount of trauma necessary to cause death. He employed a falling weight and expressed the energy input necessary to cause death in terms of its mass and the distance the object dropped. Death did not occur in the guinea pig unless the energy of the blow exceeded 2.75 kg. meters. Injection of ascorbic acid immediately after the minimum injury which would have caused 100 per cent mortality in untreated animals, allowed survival. Withholding the vitamin C injection for an hour decreased the survival 50 per cent.

As in the case of guinea pigs, it was found that rats which were traumatized at a level energy-input at which control experiments showed that death would normally ensue, survived if given ascorbic acid shortly after trauma, in a concentration of 100 mg. per kg. of body weight. (*Nutrition Reviews* 1:93, Jan. 1943).

There are several points to be considered when interpreting these laboratory results. Foremost, general conclusions gained from guinea pigs and rats cannot be extended directly to man. It is known that guinea pigs require relatively greater amounts of vitamin C than does man (Bourne, *Lancet* 1: 688, May, 1944). Furthermore, man, monkey, and the guinea pig are the only animals known to develop deficiency of vitamin C. All others apparently have the ability to synthesize it. Thus, Ungar's results in the rat-trauma studies along with the huge amounts given and the need for immediate administration suggest a pharmacologic action rather than a "nutritional" one in the experimental effects.

Nevertheless, the importance of an adequate intake of vitamin C in securing maximum healing of skin and bone wounds is now recognized, although more information is needed as to the actual amounts desirable. Crandon and his associates *(N. Eng. J. Med.* 223: 353, 1940) found in one patient, that even after three months on a diet completely deficient in vitamin C and within whom the plasma vitamin C had been nil for 44 days, a wound in the man's back apparently healed quite well as far as clinical and histological observation were concerned. The period taken for complete breakdown of wound healing on a completely deficient intake was 6 months. But we rarely have to deal with

human beings who have failed to take any vitamin C at all. Human vitamin C deficiency is more likely a small, probably "subclinical" deficiency extending over a long period. Thus, if Crandon's results are generally applicable, it would seem likely that only occasionally is a vitamin-C deficiency a factor in failure or delay of wound healing. Further evidence for this was shown by Pijoan and Lozner (*Bull. Johns Hopkins* 75: 313, 1944). They reported healing of a wound similar to that of Crandon's in 6 days in a man who had been on low vitamin C for 21 months (80 days on 5 mg. intake, remainder on 18-25 mg.). Biopsy showed no evidence of weak areas in tissue structure.

Bourne (*Lancet* 1: 688, 1944) decided to compare tensile strength of wounds in guinea pigs with their histological appearance in varying degrees of vitamin C deficiency. His experiments showed that wounds in animals which are receiving different doses of vitamin C may be similar in general histological appearance but may have very different tensile strengths. In one case, for example, two animals, one of which had been on 1 mg. of vitamin C daily for 2 weeks and the other on 2 mg., had scars which looked much alike when stained; in fact, the scar from the 1 mg. animal appeared to have healed better than that of the 2 mg. animal. The tensile strength of the first, however, was 135 g. and that of the second 320 g.

Bourne says:

> The greatest difficulty in this work is to secure a proper basis for comparison of guinea pig doses of vitamin C with human requirements. A guinea pig needs about 20 mg. of vitamin C (daily) for saturation and a human being about 30-40 mg. Daily doses of 30 mg. of vitamin C to guinea pigs failed to improve the tensile strengths of wounds over those of guinea pigs receiving 2 mg. Jones and others (*N. Eng. J. Med.* 229: 153, 1943) found no significant difference between the average tensile strengths of wounds in animals receiving 1.5 mg. of vitamin C daily and those receiving 15 mg. It is apparent therefore that saturation with vitamin C is not essential for optimal healing, at least of small wounds in the skin, but Jones and his colleagues found that maximal saturation of tissues with vitamin C helped to provide optimal resistance of the tissue to infection.

Vitamin C is likewise needed for proper bone regeneration. One of the functions of vitamin C is to allow the production of a phosphatase-impregnated bone matrix upon which bone salt is immediately deposited. As long as there is sufficient vitamin C to produce matrix, then the matrix will be calcified (Bourne. *J. Physiology* 102:319, 1943).

AMOUNTS REQUIRED FOR WOUND HEALING

Although vitamin C is certainly necessary for healing of wounds, experimental data indicate that large saturation intakes are not of especial benefit. The recommendation of Bourne (*J. Physiology* 101:327, 1942) that "patients with fractured bones should be given at least 40 mg. of vitamin C daily as long as there is the slightest doubt about the efficiency of smaller doses" seems reasonable and warranted according to present data. He states that this dosage probably applies also to regeneration of skin wounds.

> Since experimental tests have all been on small wounds, [says Bourne,] it seems possible that extensive injuries might require much more vitamin C for optimal healing. The healing of a wound is an emergency process in an animal and one would expect reserves of necessary substances in the body to be called on for this purpose. This

appears to be so for vitamin C, since Lauber and Rosenfeld have shown that if animals are on a low vitamin C diet, the organs lose most of their vitamin C when the animal is wounded and the vitamin can be demonstrated in the tissues surrounding the wound.

Because of this withdrawal of vitamin C from other tissues to fulfill the needs of the healing tissues, it appears desirable to administer vitamin C to all injured persons routinely.

VITAMIN C NEEDS OF THE ADULT

An adequate daily intake of vitamin C for the average adult should be an amount between the protective minimum of 18-25 mg. and the amount required to maintain saturation. The precise intake is largely a matter of conjecture and opinion. The recommended allowance of the Food and Nutrition Board, National Research Council, Revised 1945, is as follows:

Man	75 mg.
Woman	70 mg.
Pregnancy (latter half)	100 mg.
Lactation	150 mg.
Children, up to 12 years	
Under 1 year	30 mg.
1-3 years	35 mg.
4-6 years	50 mg.
7-9 years	60 mg.
10-12 years	75 mg.
Children over 12 years	
Girls 13-15 years	80 mg.
16-20 years	80 mg.
Boys 13-15 years	90 mg.
16-20 years	100 mg.

These allowances are sufficiently generous to allow for losses which may occur due to the manner of preparation of food, especially to its being held after preparation, exposed to the air for considerable periods of time before it is eaten; and to the variations in original vitamin content of foods.

From the standpoint of prevention of gingivitis, the recommended allowances should be ample. As previously pointed out, numerous carefully controlled studies on human beings have shown that clinically recognized scurvy does not develop in adults on a daily intake of 18-25 mg. of vitamin C. (Stamm, Macrae and Yudkin, *Brit. Med. J.* 2: 239, 1944; Pijoan and Lozner, *Bull. Johns Hopkins* 75: 303, 1944; Najjor, Holt, Rayston, *Ibid*, 315).

Furthermore, it is highly questionable that the gingival lesion is a scorbutic one on the basis of saturation tests and plasma levels. The incidence of bleeding gums was not reduced by treatment with 100-200 mg. of ascorbic acid daily over a period of three weeks by Stamm and his co-workers. Contrary clinical results are reported by Kyhos, Gordon, Kimble, and Sevringhaus *(J. Nutrition* 27: 271, 1944). They state that 12 human subjects on 25 mg. of synthetic vitamin C for 5 weeks showed definite improvement of oral tissues; and that "with a 75-100 mg. supplement, the most satisfactory response of both plasma and gum healing was obtained." In evaluating the significance of an increase in plasma ascorbic acids levels, one must keep in mind that many studies, including a recent one on 959 individuals by Darby and Milam *(Am. J. Pub.*

Health 35: 1014, 1945) show that there is no significant statistical correlation between gingivitis or bleeding gums and plasma vitamin C. Low plasma values do not necessarily indicate a scorbutic process (Pijoan and Lozner, *N. Eng. Med. J.* 231: 14, 1944), unless such values exist concomitantly with a deficiency of the white cell-platelet content. Ascorbic acid content of the white cell-platelet layer of centrifuged blood is probably the most accurate indicator of the prescorbutic status. Any fixed level of vitamin in the plasma, irrespective of how little is present, indicates a nonscorbutic process and positive body economy. A low level is suggestive of a lessened reserve.

Since no control cases were run by Kyhos and associates simultaneously with the test and, also, since the person who performed the dental examinations knew that vitamin C supplements were being given to all patients, there is, in addition, the problem of a possible bias in interpretation of clinical results or "gum healing". Without controls there was no check on possible influences of improved mouth care or other contributing factors. In other studies, as for example that of the personnel of the Royal Air Force stations (Stamm, *et al*), the examiner had no knowledge of which persons were receiving vitamin C and controls were run.

Unquestionably, some cases of bleeding gums are due to lack of sufficient vitamin C. In such instances, a therapeutic trial of 300 mg. ascorbic acid a day should bring about rapid healing in a relatively short time. Campbell and Cook *(Brit. Med. J.* 1: 360, 1941) found that most of their cases responded in four days, while 14 days was the maximum time required. They said that 100 mg. maintained their cases, although they did not report trial of lesser amounts. Their favorable results were obtained without other dental treatment such as scaling or mouthwashes. It is noteworthy that the incidence of gingivitis was very low in their experimental population, since of 505 new patients admitted to the hospital in 10 weeks, only 18 had gingivitis of such nature that the gums would bleed upon finger pressure.

FULFILLING THE VITAMIN C NEEDS OF ADULTS

No doubt a considerable number of adult Americans, whether urban or rural, are not securing a sufficient amount of vitamin C for best health. The increased cost of citrus fruits is less frequently a cause than failure to realize the need for use of those foods which contain ample amounts of vitamin C. The problem then, is essentially educational; that is, primarily along lines of motivation to change food habits. It has been repeatedly shown that nutrition knowledge itself does not modify food habits substantially (Gold, "Can Schools Change Food Habits?"—Nat. Res. Council Comm. on Food Habits, 1944; Brown, "Diets of High School Students," Univ. Minn. Press No. 4, Apr. 1945).

An individualized program directed toward changing food habits should be conducted. Proper recommendations by the dentist carry weight with the patient. In the case of vitamin C, the dentist can justifiably advise the daily use of either orange, grapefruit or tomato plus a serving of potato to ensure sufficient intake for normal needs. Together these two foodstuffs will usually furnish ascorbic acid to fulfill the standard of 70-75 mg. This, even though the vitamin C content of tomato is on the average approximately half that

of the citrus fruits and that of potatoes varies with the season. The content of autumn potatoes is about 11 mg. per 100 gm. with a 50 per cent loss through storage so that the content is reduced to 5-6 mg. by spring.

Although liberal use of citrus fruits is desirable, it is well to warn patients of possible deleterious effects from the daily use of lemon juice in water. Clinical reports suggest that many cases of sensitive teeth and resorption of exposed lingual root surfaces have their inception in this practice. The diluted solution seems more harmful to tooth structure than does the undiluted juice or than does orange or grapefruit juice undiluted taken with other food.

Foods to be recommended as dependable sources of vitamin C must have an original high content, must be commonly available to the general population, and must contain reasonably large amounts of the vitamin after preparation. Citrus fruits and tomato, fresh or canned, satisfy all these requirements. Losses of ascorbic acid in other foods (and B-complex vitamins also) in quantity cookery may be as great as 95 per cent according to Nagel and Harris (*J. Am. Diet. A.* 19: 23, 1943). In general these results have been verified and extended to include other vitamins by Heller, McCay, and Lyon (*J. Nutrition* 26: 377, 1943).

Studies conducted at one of the cafeterias feeding 1400 office workers and 1000-1400 industrial workers at the Brooklyn Navy Yard were designed to give an approximation of the losses of vitamins in large-scale cookery. Samples were collected (a) raw, or frozen (before defrosting); (b) immediately after cooking; (c) of the cooking water immediately after vegetables were cooked; and (d) of the food in the steam table when serving ended. The losses in per cent, ranged as follows: thiamin 16-64; niacin 2-61; riboflavin 22-45; ascorbic acid 27-90. Such losses demonstrate the need for more careful control of cooking and serving procedures in order that the food eaten may have a larger quota of nutrients.

With so many people depending upon "quantity-cooking" sources for their meals, proper nutritive intake can be a serious problem. Thus, although some persons may sincerely try to eat a satisfactory diet, they may fail for causes beyond their control. Practicality dictates the use of supplementary vitamins for persons who cannot regularly consume a satisfactory diet, for whatever reason.

Supplementary preparations are those which provide amounts of specific nutrients to ensure an adequate intake thereof. As a rule, formulas of supplementary preparations supply in one dose or in divided doses, the amounts of vitamins given below:

Vitamin A	4000-5000 International Units
Vitamin D	400-800 International Units
Thiamine hydrochloride	1-3 mg.
Riboflavin	2-3 mg.
Niacin Amide	10-20 mg.
Ascorbic Acid	30-100 mg.

The one vitamin least likely to require supplementary use in the United States is vitamin C (ascorbic acid). It is the only one for which a single daily average serving of commonly available foodstuff (citrus fruit or tomato) will provide at least the protective minimum.

832 LaSalle Bldg.

Notice to Members of the American Academy of Periodontology

AT THE last annual meeting held in Houston in 1941, the Sub-Committee on Scientific Investigation made the following suggestion in their report:

That the members of the Academy send to this Committee each year several complete case histories of interesting periodontal cases, including roentgenograms and photographs before and after treatment. Evaluation of a large number of these records should give much interesting information on periodontal conditions and treatment procedures.

In response to a post card sent to all members, many signified their willingness to coöperate with the Committee and case history forms were sent to those who requested them. In the meantime, the events of the past four years made it difficult for the members to prepare these histories.

The war is over, our annual meeting will be held this year, and it is the wish of the Council that this Committee continue its work. If research in dentistry and clinical investigations in the field of periodontia are to continue, it is necessary that those projects interrupted by adverse conditions should now be resumed with renewed vigor.

Therefore, we ask the members of the Academy for their coöperation by filling out the enclosed post card and sending in one case report with roentgenograms. The Case Report should be sent to the *Chairman*, Dickson G. Bell, 450 Sutter Street, San Francisco 8, California at least two months before the date of the 1946 Annual Meeting, in order that the Committee may have sufficient time to evaluate the data.

Selected Case Reports will be published in future issues of the *Journal of Periodontology*.

SUB-COMMITTEE ON
SCIENTIFIC INVESTIGATION.

American Board of Periodontology

Suggestions for Preparation of Case History—(1942 Edition)

I. DENTAL HISTORY

Patient's complaint, first manifestation of symptoms, previous treatments, including ulcerative gingivitis (Vincent's infection) and Orthodontics, results, lateral abscesses, toothaches, loose or sore teeth, why teeth were extracted, scars, caries susceptibility and age of greatest amount of decay, self-mouth and throat medication habits (mouth washes, lozenges, etc.).

II. CLINICAL-ANATOMICAL FACTORS

A. Periodontal Tissues

Gingival color, absence of stippling, inflammation, edema, atrophy, hypertrophy, hypersensitivity, hemorrhage—spontaneous or on probing, suppuration, ulcerative gingivitis, lateral abscesses, pockets, calculus, bifurcation involvement, sensitive root surfaces, severity of gingivo-periodontal disturbances.

B. Individual Teeth

Vitality, possible pulp involvement, discoloration, caries, hypoplasia, Hutchinson's notches, abrasions, erosions, improper fillings or other restorations, excessive filling material, crowns or removable appliances causing gingival inflammation, incorrect contour of restorations, faulty contacts. Note any individual defects causing gingival or periodontal disturbances.

C. Hygiene and Miscellaneous

Stains, sordes, mouth odor, food impactions, signs of toothbrush trauma on teeth or gingivae, toothbrush habits. Abnormal saliva, mouth breathing, gagging.

Mucous membrane, tongue, and sublingual area. Description of any lesion found, color, coating, indurations, ulcers, warts, leukoplakia, necrosis of tissues, scars, mucous patches.

Significant anatomical abnormalities not already noted, such as enlarged tongue, abnormal temporomandibular joint, etc.

Obvious swellings about face and neck, asymmetrics, adenopathy, scars, fistulas.

III. CLINICAL-FUNCTIONAL FACTORS

A. Occlusion

Type of malocclusion—overbite, overjet, excessive stress—cuspal and incisal interference.

Missing teeth—cause.

Teeth in infra-occlusion, tilted, migrated, rotated, extruded.

Mobility—cause.

Occlusal defects causing or tending to cause periodontal injury.

B. Necessary restorations or prosthetic appliances.

C. Existing replacements

Function, aesthetics, phonetics, retention.

D. Habits

Grinding or clenching teeth at night, biting on pipe stem, thread, etc., biting soft tissues.

Excessive prying force from biting resistant foods.

IV. ROENTGENOGRAPHIC EXAMINATION

A. Type of alveolar resorption—horizontal or vertical, root resorption, pulpless teeth, poorly filled root canals, pulp stones.
B. Condition of alveolar bone, especially any deviation from normal appearance (porotic or sclerotic areas).
C. Width of periodontal membrane space.
D. Fragments of broken roots or other foreign bodies.
E. Necrotic areas or sequestra.
F. Impacted teeth or congenital anomalies.
G. Cysts or other evidence of pathologic involvement.
H. Periapical infections—granuloma, cementoma, etc.
I. Relationship of teeth to maxillary sinus.

V. MEDICAL HISTORY AND SYSTEMIC FACTORS

A. Recent illnesses, loss of weight, loss of appetite, frequent colds, cough, bloody sputum, night sweats, excessive thirst plus increased appetite, frequent urination and loss of weight, shortness of breath and swelling of ankles, dizziness, headache, and disturbances of vision, vomiting, diarrhea, blood in stools, aches or pains in muscles and joints.
B. Occupational diseases (as lead poisoning, flour dust caries, etc.).
C. Use of drugs and intoxicants (cathartics, sleeping powders, mercurials, alcohol, tobacco, etc.).
D. Mental and psychological factors.
E. Signs and symptoms of endocrine imbalance.
F. Evidence of oral foci of infection.
G. Need for medical coöperation (as in oral tumors, soft tissue ulcers, suspected diabetes, leukemia, etc.).
H. Pre-operative precautionary measures indicated in cases of diabetes, apprehension, bleeders, novocain hypersensitivity, pregnancy, heart ailment, etc.
I. General appearance—signs suggesting ill health, nervous disorders or unfavorable temperament as indicated by uncoöperative attitude, gait, manner of speech or condition of dress, aspect of good or bad health, weight, nervous habits (of face, hands, etc.), paralysis, unhealthy skin, etc., which suggest an unfavorable prognosis.
J. Laboratory tests and consultations.
K. Nutrition History

The dietary pattern accepted by the Council on Food and Nutrition of the A.M.A. is an excellent guide for checking the patient's food habits.

Milk—2 or more glasses daily for adults; 3 or more daily for children.

Vegetables—2 or more servings daily besides potato; 1 raw; green and yellow often.

Fruits—2 or more servings daily; 1 citrus fruit or tomato.

Eggs—3 to 5 a week; 1 daily preferred.

Meat, cheese, fish, or legumes—1 or more servings daily.

Cereal or bread—most of it whole grain or "enriched."

Butter or fortified oleo—2 or more tablespoons daily.

VI. DIAGNOSIS

Summary statement of periodontal disease with analysis of causes.

VII. PROGNOSIS

VIII. THERAPY

Consideration of treatment for each defect noted which is related to present or possible future periodontal disease.

IX. EPICRISIS

Results of treatment (local, systemic, aesthetic, functional).

Records of treatments with dates and reactions to treatments.

Post-Operative Instructions

CASE HISTORY RECORD

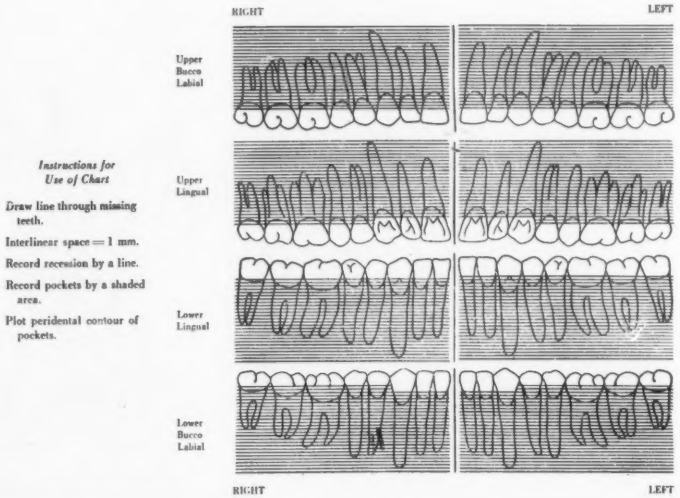
(Record should be typewritten)

Applicant's Name _____ Address _____

Patient's Name _____ Occupation _____ Sex _____
Age _____ Weight _____ Height _____ Marital Status _____
Birthplace _____ Nationality _____
Date and place of last treatment _____
Model Date _____ Photograph Dates _____ Roentgenogram Dates _____

Summary of:

I. DENTAL HISTORY:



Instructions for Use of Chart

Draw line through missing teeth.

Interlinear space = 1 mm.

Record recession by a line.

Record pockets by a shaded area.

Plot peridental contour of pockets.

Appearance of gingival tissues:
Bright pink_____|_____ Red_____|_____ Bluish_____|_____
Atrophic_____|_____ Hypertrophic_____|_____ Ulcerative_____|_____
Edematous_____|_____ Hemorrhagic_____|_____
Suppuration:_____|_____ Lateral abscesses_____|_____
Calculus: Supragingival_____|_____ Subgingival_____|_____
Bifurcation involvement:_____|_____ Sensitive root surfaces_____|_____
Abrasions_____|_____ Erosions_____|_____ Toothbrush trauma_____|_____
Gingival inflammation due to improper restorations:_____|_____
Incorrect contour of restorations or faulty contacts from any cause:
Lack of contact _____|_____ Embrasures: Closed _____|_____ Too wide_____|_____
Hygiene and Miscellaneous:

III. CLINICAL-FUNCTIONAL FINDINGS:

Describe occlusal defects:

Missing teeth:
Congenitally _____|_____ Extracted_____|_____ Unerupted_____|_____
Anomaly of position of teeth: Infra-occlusion_____|_____ Tilted_____|_____
Migrated_____|_____ Rotated_____|_____ Extruded_____|_____

Mobility:
1st degree—detectable under stress_____|_____
2nd degree—move rather easily between two forefingers_____|_____
3rd degree—very marked looseness_____|_____

Miscellaneous:

RIGHT LEFT

IV. ROENTGENOGRAPHIC FINDINGS:

Type of alveolar resorption: Horizontal atrophy---|--- Vertical atrophy---|---

Root resorption:----------|---------- Pulp stones----------|----------

Pulpless teeth:----------|---------- Granuloma----------|----------

Condition of bone structure: Normal----|---- Sclerotic----|---- Porotic----|----

Widening of periodontal space:----------|----------

Length of roots (as it affects prognosis):----------|----------

Miscellaneous:

V. MEDICAL HISTORY:

Dietary summary and recommendations:

VI. DIAGNOSIS:

VII. PROGNOSIS:

VIII. THERAPY:

a. Type of local therapy:

b. Correction of functional disturbances:

c. Correction of systemic disturbances:

IX. EPICRISIS:

Results of therapy:

Photographs of case before and after treatment:

Editorials

The Need for Optimism

THE POST-WAR reaction to the tension of the past four years is seemingly worse than the war strain itself. There is often a depression of spirits after unusual excitement or tension. But that we were unprepared for the current excessive depression of mind is frequently acknowledged. The pessimistic tenor of the material one reads in the daily press and current popular magazines, and hears over the radio from commentators, politicians, pulpit orators, and scientists, does not furnish much cheer. The dangers of the atomic bomb, the hatred and jealousies of races and of nations, and the threats of epidemics, a crime wave, country-wide strikes, and socialism are discussed. The keynote of an antidote for all this was struck by a popular song writer who advised to "accentuate the positive". Let us have a try at it.

Granted that we do have strikes of unprecedented proportions—the recent war was also without parallel, the repercussion from which was bound to produce upheavals of various kinds. True we have the atomic bomb on our hands, but we can *demand* that the United Nations Organization find the solution for that problem. The work on the atomic bomb has already resulted in benefits to the human race, for one instance, in the treatment of cancer. While we have not learned to love our neighbors as well as ourselves, we are definitely improving in that respect. We have had epidemics before, but now we have a greater number of measures for combating disease.

A recent medical journal quoted George Gissing in the following: "It is because nations tend to stupidity and baseness that mankind moves so slowly; it is because individuals have a capacity for better things that it moves at all". This is a challenge to the individual.

There never was a time when there was more serious thinking being done, as conversation with almost any acquaintance will verify. This is encouraging. There is an earnest desire of former service men and women, as well as adult civilians, for further education or special training in some useful occupation. This is an especially desirable trend.

Dentistry is beginning to be recognized as being necessary to the health of the nation. When the cartoonist and the humorist use the dentist as a subject, which they are doing frequently of late, we know that, that is the first step in developing the appreciation of the public for dentistry. A conservative number of dental patients are beginning to be aware of the difficulties and skill required in dental operations and technics. Dentists have never before had such an opportunity to deserve and to get proper recognition.

We can be justly proud of the many officers of dental societies who carried on during the war without any glory, hard work being their chief reward. Pages 42 and 43 carry the names of many men and women dentists who have expressed in tangible form their interest in the advancement of dental journal-

ism. By this gesture they have not only given material assistance to an ideal, but have furnished inspiration for many now living and for those who come after. It is a positive demonstration of an appreciation for the best things in life.

Dental educators are thinking seriously about the future of dentistry. More dentists are now studying the trends in the social welfare of the people from a broad viewpoint.

Dentistry has a part to play in building the long promised better world and all trends of dental thought lead to the conclusion that she will courageously and effectually assume her responsibilities.

Advanced Education in the Sciences Related to Periodontology

IT IS questionable if many periodontists are satisfied with their understanding of the medical and related sciences. Because periodontia is more dependent upon bodily conditions for its results in treatment than any other part of dental practice, additional knowledge of these subjects is necessary. The cementum, the periodontal membrane, and the alveolar bone are the last links in the preservation of the teeth in the mouth. Even if the pulps of the teeth have been destroyed, but enough of the periodontium is in a state of health, the teeth can still serve their intended purpose. This is not to argue for the retention of pulpless teeth without the most thorough diagnosis and skillful treatment, but to emphasize the close dependency of tooth function upon periodontal health.

Many serious systemic conditions manifest themselves first, symptomatically, in the mouth. The periodontist can wisely extend his usefulness by becoming familiar with these diseases, and others resulting from focal infection, the latter often traceable to a primary focus in the mouth. Among the evidences that point to a need for further education in medical subjects is the "Seminar in Dental Medicine" given for several years, in California. It continues for six days and is well attended.

For five years a three-day Seminar has been held at the W. K. Kellogg Foundation Institute in Ann Arbor for members of the American Academy of Periodontology. Unfortunately, the living accommodations there are very restricted and for this reason the attendance has had to be limited to a small number.

Another Course of Lectures is being arranged this year and will be given April 29-May 1 inclusive. The program will be printed in the April, 1946 issue of this Journal. Reservations can be accepted for Academy members only, and the number is limited to thirty.

It must be emphasized that this Seminar should not be confused with a post-graduate course in periodontia, which it is not, or with a dental meeting. It is planned only to give an opportunity for serious advanced study of the sciences related to periodontology and is conducted with the strict schedule and formality of university rules.

—G.R.S.

BOOK REVIEW

Dental Chronology. By Hermann Prinz, A.M., D.D.S., M.D., Sc.D., Dr.Med.Dent. Emeritus Professor of Pharmacology and Lecturer, The Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania. Philadelphia, Lea and Febiger, 1945. 189 pp., 97 engravings.

Readers of dental literature are familiar with the writings of Dr. Hermann Prinz and the announcement of a new book by him is quite naturally greeted with enthusiasm. The author of *Dental Chronology* emphasizes that the book is a "guide" to the study of dental history and nothing more. One is nevertheless impressed with the amount of valuable information contained within the pages of so small a volume.

The book is divided into three parts: I. First Period—Antiquity—x-1000. II. Second Period—The Middle Ages—1000-1728. III. Third Period—Modern Times—1728-x. In addition there are interesting tables and short notes and a bibliography of dental history. The general make-up of *Dental Chronology* will intrigue any dental student or tired dental practitioner, with its large type and interesting illustrations surrounded by ample page space. The effect is most pleasing.

Among the events listed as having an influence on the advancement of dentistry the author has mentioned the founding of various dental schools and the organization of several national dental societies. One of the latter, the American Society of Orthodontists, organized in 1900 is listed, but no mention is made of the American Academy of Periodontology which was organized in 1914, and has done more than any other group to promote interest in the prevention and treatment of periodontal disease. Since 1930, it has published the *Journal of Periodontology* largely financing it with funds from its treasury. Although reference is made to the introduction of the term "paradontosis" by a European (1923) to "serve as a substitute" for pyorrhea alveolaris, nothing is stated with regard to the nomenclature adopted by the Academy, and now quite generally used in this country. While one can understand the necessary omission of many important events in a "guide" to dental history, the recording of many minor items, such as the introduction of "iodoglycerole into dental practice" (1912) and an " 'Epithelial Solvent' for the destruction of the epithelial lining of the pockets in pyorrhea alveolaris" (1926), leaves the reader puzzled about the before mentioned event which was overlooked. Readers of *Dental Chronology* should make an entry on one of the blank pages, thoughtfully included in the book for the purpose

(Continued on page 43)

In Memoriam

ARTHUR J. COTTRELL

October 31, 1945

PAUL R. STILLMAN

December 15, 1945

PAUL ROSCOE STILLMAN

1871 — 1945

IN THE PASSING on December 15, 1945 of Paul Roscoe Stillman the American Academy of Periodontology has lost one of its most eminent members. His contributions to the Academy itself as well as to dentistry and the science and art of periodontology were so great and so varied as to be difficult of appraisal. Paul Stillman suffered a stroke more than ten years ago and shortly after moved to Florida in a vain effort to regain his health. While keeping up a lively interest in dental activities, especially in the field of education, and producing several notable papers in that period, he was unable to return to active practice.

Probably no one dentist has done more than he to bring periodontia to the dental profession, to make the profession conscious of its responsibility and opportunity in this field. Through the force of his remarkable personality, his great popularity, and wide acquaintance, he impressed on the entire profession, both general practitioners and members of other budding specialties, the fundamental part periodontal considerations play in the work of all.

Of direct and readily visualized importance was his dramatization of the etiologic factor of occlusal overstress by giving it the name "traumatic occlusion." While his proposal of this term, together with his frequent and effective demonstrations of the detection and correction of the condition, by no means represents his total contribution in this field, his reputation might well rest on that achievement alone. However, he demonstrated his grasp of the entire range of periodontology in his writings, most notable of which was the *Textbook of Clinical Periodontia*, in the writing of which I was fortunate to be co-author. As a clinician he was preëminent, exhibiting unusual skill but more especially displaying to a remarkable degree the ability to interest and stimulate his audience.

Paul was a living demonstration of the saying that the way to have good friends is to be one, and because he was such a good friend his friends were legion. I have known no one in the profession more generous, more desirous of being helpful to others than Paul Stillman. It is impossible to estimate or to state how much his friendship meant to me.

It is not my intention nor would Paul want this tribute to be cast in serious vein only. No one enjoyed more than he the lighter moments of life or was more ready and able to contribute to them. All who knew him well will recall incidents that will bring smiles and that is as he would want it.

To the American Academy of Periodontology Paul contributed much, not only as Councillor and President in its early years, but by impressing on the profession at large the solidity and stability of its foundations and the part the Academy would inevitably play in the advancement of dental science.

Paul R. Stillman was born in Greenwich, N. Y., June 4, 1871. He was graduated from the Baltimore College of Dental Surgery in 1899. After practicing for a short time in Greenwich he established a practice in Brooklyn and then in New York City. He became particularly well known as a clinician during the period from 1916 to 1920 when he gave many postgraduate courses under the auspices of Columbia University. In 1925 he became Clinical Professor of Periodontia in the New York University College of Dentistry. His activities were not limited to practice and teaching, however. He was one of the charter members of the International Association for Dental Research. He was at one time active in the New York National Guard as a dental surgeon and was for some years a member of the New York City Mayor's Dental Advisory Committee. He was also active in the American Arbitration Society. He was a member of Psi Omega and Omicron Kappa Upsilon.

JOHN OPPIE MCCALL

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Wall, B.	10.00	TOTAL	$2496.00

BOOK REVIEW— *(Continued from page 40)*

of making notes—"American Academy of Periodontology, organized May, 1914 at Cleveland, Ohio. Publishers of the Journal of Periodontology" [Not Journal of Periodontia, *See* p. 156].

Other errors that should be corrected are to be found on p. 126, viz., "1886—Charles H. Land introduced the all-porcelain jacket crown," and on p. 127, "1889—(a) All-porcelain jacket-crown was patented by C. H. Land." In a pamphlet titled "Land System of Dental Practice," temporarily loaned to this reviewer, and copyrighted 1893 by Charles H. Land, seven patents are listed and his "system of dental practice" is described in detail and well illustrated. He makes no mention of an "all-porcelain jacket-crown," but uses the terms "metallic enamel jackets" "a metal jacket . . . a thin coat and veneer of porcelain," "metallic enamel caps," "enameled caps of metal coated with porcelain," "a coating of platinum made to fit the outside of the teeth, after which the anterior surface is coated with a porcelain enamel front."

In the *Dental Cosmos*, April, 1891, Dr. W. A. Capon of Philadelphia describes the method patented and used by Dr. C. H. Land for making the so-called "jacket" crown. A platinum tube was used and closely fitted to the neck of the tooth. The finished crown had a palatine surface of platinum and the labial surface of porcelain was fused upon the platinum. The "all-porcelain jacket-crown" technique was not developed until 1903, when it was first demonstrated.*

—G.R.S.

*See *D. Cosmos* 45:615-620. Aug. 1903.
Items of Interest 26:401-410. June 1904.

AMERICAN ACADEMY OF PERIODONTOLOGY

Three-Day

Post-Graduate Lecture Course

W. K. Kellogg Foundation Institute

APRIL 29, 30, and MAY 1, 1946

ANN ARBOR, MICHIGAN

For Members

Discussion of "Periodontia Procedure"

*(Continued from October, 1945 issue)**

WALTER LEABO

Shreveport, La.

I regret that my discussion is too late for publication, but I wish to confirm what seems to me to be the most concise and complete statement of periodontal practice and procedure that has come to my attention. So far as I am able to evaluate the statements there are no fundamental principles mentioned either in etiology or treatment, to which exception can be taken in the light of previous research and the practical experience of the majority of operators extending back for many years.

The greatest need in dentistry today is an acceptance of just such principles as are given in the "Initial Statement," and a very definite program for teaching to undergraduates the absolute necessity of every dentist being thoroughly grounded in the prevention and treatment of periodontal disease.

The profession generally, and this unfortunately frequently applies to the college student and recent graduate, seems to hold to the belief that only by some spectacular operation can anything worthwhile be done for periodontal lesions. Observation in my section of the country impresses me with the fact that an air of complete pessimism prevails where periodontal disease is concerned. This is a deplorable state of affairs in a profession which should have as its objective and ideal the saving of teeth. We still seem to be turning out dentists whose sole aim appears to be the construction of a good inlay, crown, bridge and finally a denture. Please understand that I am not decrying this type of skill since it is most essential, but ultimately of little value if the health of the foundation tissues is neglected. An acceptance of fundamental principles, better and more uniform undergraduate education is the only answer.

As you may know I have always been an advocate of subgingival curettage as the very foundation of periodontal treatment, without which all other operative methods of treatment or prevention are bound to fail, and evidently a procedure which cannot be relegated to a hygienist if even good prevention is to be practiced. The "Initial Statement" as I interpret it conveys emphatically the same principle. It is hard to understand, looking back over thirty years of practice, why our profession has made so little progress in the teaching of periodontology in our colleges, considering it is a subject of such vital importance in accomplishing the ideal toward which we like to think we are striving, the saving of teeth and the elimination of dental infection.

Probably the greatest factor, as I see it, in inadequate undergraduate instruction is the confusion generally existing concerning etiology and operative methods. I am speaking now of the rank and file of practitioners. Of all the groups within organized dentistry the American Academy of Periodontology is in the position of being able to dispel, to a large extent, this confusion of ideas and theories, and to formulate and insist upon a more or less standard system of teaching. I think the "Initial Statement" is a real and practical contribution to this end. If every undergraduate today could be properly impressed with the necessity of mastering the principles so simply expressed, what a difference it would make in the practice of dentistry within the next ten years!

While there may be differences of opinion as to specific techniques in instrumentation, correction of trauma, tooth brushing and other home care procedures, premedication, etc., I feel these are factors of minor importance as compared to the fundamental issues expressed in the "Initial Statement."

The next "In Our Opinion" discussion will appear in the April, 1946 issue.
Subject: "Occlusal Equilibration."

*Because of sudden illness, Dr. Leabo was unable to get his formal discussion in on time for publication in the October issue. However, in a letter to Dr. Harold J. Leonard later, he discussed the subject quite fully. With the consent of Drs. Leabo and Leonard extracts from the letter are here published.—ED.

ABSTRACTS OF CURRENT DENTAL LITERATURE

(Continued from 16(4):157. Oct. 1945)

(e) Symptomatology and Diagnosis

(Continued from 16(4):157. Oct. 1945)

380—Dodd, Katherine, Leland M. Johnston and G. John Buddingh: **Herpetic stomatitis.** *J. of Pediatrics* **12(1):** 95-101. Jan. 1938.

A type of stomatitis which the authors believed to be a clinical entity was observed in 88 child patients at Vanderbilt Hospital. This was apparently identical with what has hitherto been known as aphthous stomatitis, the misnomer probably being due to the confusion of terms in text books and ignorance as to etiology. Of the many types of stomatitis in children, only in thrush has an etiologic agent been determined. In this series there were children of all ages but most were between one and three years of age, and it was probably contagious since often there was more than one case in a family. The disease seemed to be self-limiting, lasting from a few days to two weeks. The onset was sudden with illness and fever, general malaise and loss of appetite, oral lesions appearing with the fever or 24 hours later. First there were reddened blisters which soon formed ulcers, shortly covered by a yellowish white membrane quite firm to the touch. In every instance there was in addition to lesions on tongue, inside of lips, and buccal and lingual mucous membrane, inflammation of the gingivæ with ready bleeding accompanied by soreness and pain on eating. A few showed herpetic lesions of lips and skin. Seven had other diseases besides the stomatitis. Smears from mouth and gums showed no predominating organisms. Twelve of these cases were investigated experimentally by inoculations of material from the lesions into the cornea of the rabbit, and typical herpes simplex lesions were produced, results proving conclusively that this type of stomatitis is a definite pathologic entity with the virus of herpes simplex as the etiologic agent. It should thus be properly designated as "Herpetic Stomatitis." [Apropos of this article, I have recently seen a typical case of herpetic stomatitis in an adult.]

—Celia Rich

381—Johnson, E. W.: **Tuberculous gingivitis with report of case.** *J. Kansas, M. Soc.* **46:**42, 1945.

Eighteen months before admission a 33 year old woman had a small ulcer on the gum above the maxillary left lateral incisor. Therapy of six months' duration was unsuccessful, the lesion spreading over the gums of the upper and lower jaws and also on the palate. The patient complained of sore gums, sore throat and hoarseness. Oral examination showed small areas of granulomatous ulceration on the gums. There was bilateral anterior and posterior cervical adenopathy. The diagnosis of tuberculosis was made by microscopic examination and guinea pig inoculation of material from the gingivae and cervical glands. Treatment consisted of bed rest, high caloric and high vitamin diet and other general measures. X-ray therapy was also used. Two years after the patient was first seen, she was considered to be clinically well.

—H. M. Goldman

382—Robertson, H. F., and Donovan, T. J.: **Erythema multiforme exudativium or bullosum.** *Military Surgeon.* **99:**259, 1945.

Erythema multiforme bullosum occurs in young men of army age during the winter and early summer months, and is often recurrent. The actual etiologic agents are unknown. Among those suspected are: Vincent's infection, streptococci, staphylococci and anaphylactic reactions. The sensitizing factors are believed to result mainly from drugs such as salicylates, arsenic, phenolphthalein, halogens, and barbiturates. The onset is abrupt with a temperature of 99° F. to 104° F., headache, malaise, sore mouth, and throat. Vesicles may appear on the lips, tongue, buccal mucosa, nose, urethra, and conjunctivae. Stomatitis is an early symptom with profuse salivation. Balanitis may be a complicating lesion. A skin eruption of the typical iris or cocade

type may or may not occur. If present, the extremities and trunk are favored and the onset may precede or follow the mucous membrane lesions. Lymph node enlargement is usually not present. Bacteriologic studies of the blood and secretions as well as the blood count are not revealing. The most dangerous complication is conjunctivitis of the catarrhal, purulent or pseudomembranous types. This may result in corneal ulceration with perforation or diffuse capacities and total loss of vision. Staphylococcus aureus has been reported as a secondary conjunctival invader. The early administration of oral and local sulfathiazole or sulfadiazine is suggested as a prophylactic measure. The association of Vincent's infection in a number of cases suggested to Chick the use of intravenous and topical anti-spirochetal therapy.

The accepted treatment is essentially symptomatic. Bed rest, adequate nutritional balance, and careful elimination of infectious or allergic agents constitute the main therapeutic procedures. Sodium salicylate, quinine, and potassium iodide have been recommended. It is the authors' opinion these may prove more detrimental than beneficial. The patient's comfort may be enhanced by the cutaneous application of antiseptic wet dressings. A painful stomatitis is aided by the use of gummy emulsion (Pillsbury).

The differential diagnosis includes pemphigus, human foot and mouth disease, erythema nodosum, dermatitis herpetiformis, and epidermolysis bullosa. Erythema multiforme bullosum is noted by its tendency to recur, occurrence in the young male, vesiculopapular skin eruption with pseudomembranous inflammation of mucous membranes, systemic disturbance, and mild or severe ocular manifestations. It should not be mistaken for Vincent's infection or varicella.

—*H.M.G.*

(f) Treatment

(Continued from 16(2):73. Apr. 1945)

695—Barach, Alvan L., Bettina Garthwaite, Enid T. Oppenheimer, Joyce Forman, and Helen Osburg: **Oral administration of penicillin.** *Science* **102(2645)**:247-250, Sept. 7, 1945.

Authors' Summary: A convenient, practical and effective procedure of administering penicillin by mouth consists of mixing the drug with one tablespoonful of aluminum hydroxide. Substantially higher blood levels are obtained following a 50,000 and a 100,000 unit dose of penicillin with this method than after administration of the drug dissolved in water, either with or without previous ingestion of a mild antacid, such as aluminum hydroxide. Absorption of penicillin on aluminum hydroxide appears to exert a local protective effect against that portion of gastric acid with which it comes into contact.

696—Fox, N., R. B. Kesel, E. R. Neary, and R. H. Herbine: **Effect of sulfathiazole in chewing gum in certain oropharyngeal infections.** *Archives of Otolaryngology.* **41**:247-318. April, 1945. Abstract in *J.A.M.A.* **128(10)**: 763-64. July 7, 1945.

Fox and his collaborators point out that effective topical application of sulfonamides to diseased oropharyngeal tissues has been difficult because certain factors prevented the maintenance of a satisfactory concentration of the drug. The smooth oral mucosa is continually washed by saliva and does not lend itself favorably to treatment by powders, ointments or solutions. Beneficial results from the local application of sulfonamide drugs in the mouth, have been reported, but they occurred in such areas as the alveolus of an extracted tooth or a cavity in the maxilla or mandible where the drug was sheltered from swallowing and salivary dilution. The authors decided to use chewing gum as a vehicle. On the basis of their studies on this medium, they arrive at the following conclusions: 1. Chewing of gum appears to be a mouth cleansing process. 2. From the standpoint of minimal systemic toxicity and maximal local anti-bacterial potency, sulfathiazole in chewing gum seems to be the preferable local chemotherapy for infections of oral and pharyngeal mucosa which are susceptible to sulfonamide compounds. 3. Best clinical results are obtained in those conditions of the mouth and pharynx in which the beta hemolytic streptococcus is the preponderant etiologic organism.

—*Copy*

697—Keefer, Chester S., Robert P. Herwick, Walton Van Winkle, and Lawrence E. Putnam: **New dosage forms of penicillin, indications, etc. (Recommendations of U. S. Food and Drug Administration.)** *J. A. M. A.* **128(16)**:1161-1164. Aug. 18, 1945.

In listing the forms, indications, and dosage of penicillin products, penicillin troches are said to be useful in Vincent's infection, dissolving one in mouth two or three times daily between meals. If the troches are labelled "masticatory" they are to be chewed at the rate of two to three troches per day or in severe cases each hour or two. Parenterally penicillin is indicated in all staphylococcic infections with and without bacteremia, and Vincent's infections. If sensitization occurs discontinue therapy. —*C. R.*

698—Lewis, Leon: **Mercury poisoning in tungsten-molybednum rod and wire manufacturing industry.** *J.A.M.A.* **129(2)**:123-129. Sept. 8, 1945.

Mercury is utilized extensively in the manufacture of certain metal products used in the electrical and radio industries. In such factories great care is necessary to prevent mercurialism. Fine distinction is drawn between acute and chronic mercurialism and cases of both types are described. Acute industrial mercurialism is almost wholly a disease of the oral mucous membrane though there may be systemic symptoms sufficient to necessitate hospitalization. Associated spirochetal infections are to be suspected and treatment instituted. As a preventive measure, prospective employees should have dental examination since the presence of chronic gingivitis, deep pockets, pronounced malocclusion, and neglected caries, all increase susceptibility to mercurialism and should bar workmen from these jobs. A general, monthly recheck is recommended. Careful sanitary engineering and housekeeping to prevent accumulations of mercury, and good ventilation are most desirable prophylactic measures. Early chronic mercurialism frequently responds favorably to removal from exposure and this is a first essential in treatment. Later chronic stages involve the nervous system with frequently unfavorable prognosis, and the treatment is largely empirical—that is symptomatic and supportive. —*C. R.*

699—MacGregor, A. B., and Long, D. A.: **Penicillin pastilles in the treatment of acute ulceration gingivo-stomatitis.** *Brit. Dent. J.* **78**:33, 1945.

Acute ulcerative gingivo-stomatitis (Vincent's infection) was rapidly and effectively treated with penicillin pastilles containing 500 units each. Patients continuously dissolved the pastilles in the buccal sulcus without chewing or sucking. In 24 hours the patients were asymptomatic and fetor was gone and in 48 hours the acute gingival inflammation and sloughing disappeared. Within 5 days, improvement of all cases was such that treatment could be stopped. —*H.M.G.*

700—Pearce, William F. and John B. McDonald: **Treatment of ambulatory patients with penicillin sodium.** *J.A.M.A.* **128(5)**:342-44. June 2, 1945.

The study included 50 men between 18 and 35, all ambulatory cases, with spirochetal infections. Of these, 21 had Vincent's stomatitis with or without angina. All were treated with intra-muscular injections of penicillin sodium in doses of 10,000 Oxford units at two hour intervals. Elimination of all symptoms and eradication of the causative organisms was accomplished within 24 hours after therapy was begun. There were no untoward reactions of importance from the penicillin. Some of the patients were given hydrogen peroxide mouth washes following penicillin, and some cases observed in the dental clinic were treated to establish proper drainage. It is especially emphasized that satisfactory results in ulcerative stomatitis are dependent on elimination of unhygienic dental conditions. This added treatment was not found necessary in Vincent's angina. —*C. R.*

701—Schuessler, C. F., Fairchild, J. M., and Stransky, I. M.: **Penicillin in the treatment of Vincent's infection.** *J.A.D.A.* **32**:551, 1945.

A series of persons with Vincent's infection treated locally with an aqueous solution of penicillin, appeared to respond more favorably than a similar group treated by other methods.

Forty-seven patients were treated with a penicillin mouth wash for an average of 3.4

days, receiving an average of 170,000 units of penicillin. There were 4.3 per cent failures. Of 32 cases treated with topical application and spray for an average of two days using slightly less than 120,000 units of penicillin, 3.1 per cent were failures. In the control group, patients using the hydrogen peroxide mouth wash, and neoarsphenamine glycerin mouth wash failed to respond to an average treatment of seven and four days respectively. When further treatment with the penicillin mouth wash was instituted, the lesions cleared up within 48 hours in all cases. Of 36 cases treated with chromic acid for an average of 8½ days, seven were classed as failures.

The authors advocate corrective dental care, including calculus removal and prophylaxis, at the earliest possible date after treatment to insure a permanent result.

—*H. M. G.*

702—Schwartz, B. M.: **Effectiveness of penicillin in the treatment of Vincent's angina.** *J.A.M.A.* **128(10):** 704-706. July 7, 1945.

In this study of 14 cases of Vincent's *angina* of tonsils, three associated with mild gingivitis, penicillin was found of definite value in every case. True evaluation of this chemotherapy for Vincent's *gingivitis* is difficult as this is frequently re-current and often associated with dental defects and faulty nutrition, especially vitamin deficiency. The penicillin was administered intramuscularly in 20,000 unit doses three times a day for five treatments. In every case subjective symptoms were alleviated in 24 hours and eliminated in 48 hours.

—*C. R.*

703—Seeberg, Victor P. and Morris F. Collen: **Calcium carbonate as an antacid for oral penicillin.** *Science* **102 (2544):**225-226. Aug. 31, 1945.

Various substances were administered with calcium penicillin to determine which would best overcome the effects of gastric acidity when drug was taken by the oral route. Calcium carbonate yielded the best results. Among others tested were dry sodium phosphate buffer, aluminum hydroxide, magnesium trisilicate or magnesium hydroxide. Calcium carbonate being a "non-systemic" antacid should prove safer to administer repeatedly than some of the buffer salts that are soluble.

—*C. R.*

704—Shallenberger, Paul L., Earl R. Denny, and Harold D. Pyle: **The use of penicillin in Vincent's angina.** *J.A.M.A.* **128(10):**706-710. July 7, 1945.

Mahoney first demonstrated the spirocheticidal effect of penicillin in 1943, and other investigators including authors of this article have previously verified his findings. Here they present a careful and scientific study of the effects of penicillin as compared with other methods of treatment in a small series of patients with Vincent's infection. They demonstrated that penicillin is remarkably effective in these cases, the bacterial symbiosis of Vincent being peculiarly sensitive to this material and more rapidly affected by it than by other advocated methods, viz., chromic acid, sodium perborate, peroxide of hydrogen, silver nitrate, mapharsen, and sulfadiazine. Penicillin was applied locally, 250 to 500 units per cubic centimeter four times daily, and in two patients it was administered intramuscularly—eight doses of 15,000 units each at three hour intervals. Clinical and bacterial response to both kinds of penicillin therapy was prompt and in some cases spectacular. The results warrant a careful trial of this method of treatment.

—*C. R.*

705—Woodward, Fletcher D. and Thomas Holt: **Local use of penicillin. Results of treatment.** *J.A.M.A.* **129(9):**589-92. Oct. 29, 1945.

In a three months study to determine the value of the local use of penicillin in infections of ear, nose, throat, and mouth, three different forms of the drug were used. (1) Solutions of the sodium salt in isotonic sodium chloride solutions. (2) A base of a water soluble jelly. (3) Lozenges of an inert base 800 units per lozenge. Among the patients were eight with proved Vincent's infection of the gingivae which were treated by frequent use of the lozenges. All were improved in three to five days, and even severe painful symptoms of gingivo-stomatitis were promptly relieved. Proper medical and surgical treatment was also employed.

—*C. R.*